JUNE

THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION

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Mouth Infections and General Health—*Abt*
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FRANK T. BREENE, D.D.S., M.D., Iowa City, Iowa.
President Elect, American Association of Dental Schools

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Original Communications

CONTRIBUTION TO THE HISTOLOGY OF THE DENTAL PULP AND PERIODONTAL MEMBRANE, WITH SPECIAL REFERENCE TO THE CELLS OF "DEFENSE" OF THESE TISSUES*

By BALINT ORBAN, M.D., Chicago, Ill.

DENTAL research workers have been somewhat slow in studying the developments of general histology in the field of connective tissue and blood. It is known that connective tissues react to bacterial, mechanical and chemical irritations in practically the same way; namely, with inflammation. Generally, we speak of inflammation if we find a round-cell infiltration or a development of granulation tissue. However, we have not gone into a deeper study of the kinds of cells that take part in the inflammation, what happens to these cells or in what ways the organism protects itself against the injurious factor.

Only by acknowledging the developments in general histology, using proper methods in the preparation of our specimens and correlating our findings with the knowledge of general histologic investigators can these problems be properly studied.

In descriptions of the normal pulp, we are offered the information that the pulp tissue is suggestive of the embryonic; for it consists of young starlike connective-tissue cells, as well as of fibrous and somewhat mucoid intercellular substance. Blood vessels, nerve fibers and lymphatics are always described as well as the odontoblasts as the producers of dentin.

This is practically all that we find in the present descriptions pertaining to the cellular elements of the pulp. A few recent German books on dental pathology mention the presence of histiocytes. They have been recognized only in experiments on animals and have never been shown in the human pulp. Their function here has also never been intelligently

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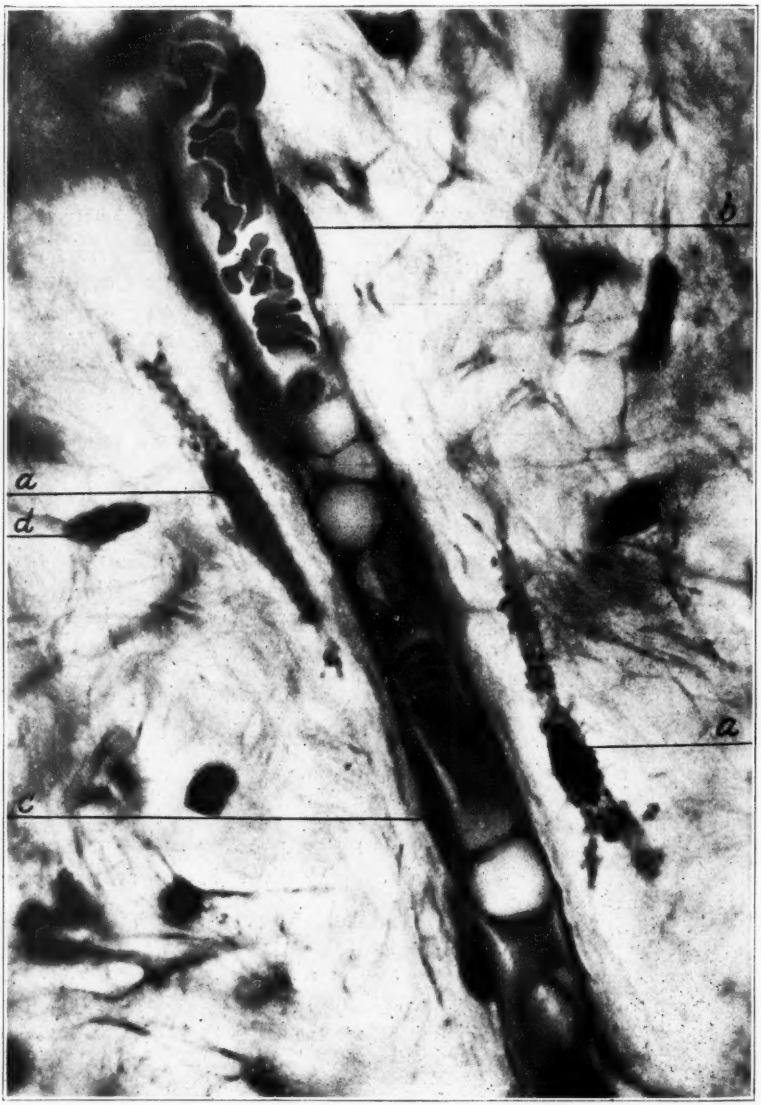

Fig. 1.—Normal human pulp. *a*, resting-wandering cells (histiocytes). *b*, undifferentiated mesenchyme cell. *c*, endothelial cell. *d*, pulp cell (fibroblast). (× 1,300.)

discussed. In the descriptions of inflammatory reactions of the pulp and periodontal tissues, we sometimes read of phagocytes, macrophages, swelling of the endothelial cells, mobilization and wandering of cells lining or closely surrounding the capillaries. None of these descriptions has been sufficiently explained, and they are therefore not clear to the reader, and most probably not even to the writer.

In the early summer of 1928, I had the privilege of making photomicrographs for the eminent histologist Alexander A. Maximow. The specimens photographed depicted the development of the cells concerned in inflammatory reactions. The paper containing these photographs was his last work, left unfinished by his tragic and sudden death. It has now been finished by his assistant and successor, William Bloom, and it will shortly appear in Ziegler's *Beiträge,* in German. While working for Maximow, I had the opportunity of discussing these problems with him, and obtained much information. Following his advice, I began to use new methods of fixing and staining dental specimens. Extracted normal as well as diseased teeth were fixed in Zenker-formol, as advocated.[1] For staining the specimen, Delafield's hematoxylin in a very weak solution was used for twelve hours, and afterwards, eosin-azure II, also according to Maximow.[2]

It was not until several months after the death of Maximow that my first specimens were ready for study. I have since consulted Dr. Bloom, as to these specimens, and I should like to express my sincere appreciation of his many valuable suggestions.

It is not my aim to enter into a discussion of the development of the different cells. That is the problem for the general histologists. Our specimens are not sufficient to solve their problems. We have to try to interpret our material according to the findings of the men who spend their lives in solving just these problems.

The bibliography on these questions is so large that it is impossible to make reference to all papers. The most extensive bibliography on papers on this subject in all languages can be found in the contribution of Maximow to the textbook of Möllendorff. Everyone who intends to study this subject further should refer to these works. Several of the most important contributions are listed at the end of this paper.

In the accompanying photomicrographs (Figs. 1-5), we shall see the cells of defense in the tissue of the pulp. The specimens are all of human teeth, and the pictures of the pulp are of normal, healthy teeth of adult individuals.

In Figure 1, we see crossing the field a capillary in which some of the red blood corpuscles are irregularly arranged, and the others piled up like coins. The endothelial cells form the wall of the vessels. The pulp tissue shows the finely fibrillated ground substance in which cells are embedded. Some of the cells are star-shaped like young connective-tissue cells; others are elongated like fibroblasts elsewhere in the loose connective tissue of the body. Some of the fibers are the protoplasmic processes of the cells, as at *d;* others are collagen-

1. A fresh mixture of 90 c.c. of Zenker's stock solution (potassium bichromate, 2.5 gm., corrosive mercuric chlorid, 5.0 gm., water 100 c.c.) and 10 c.c. of 10 per cent neutral formalin (40 per cent solution of formaldehyd, U. S. P.).

2. Two stock solutions are kept ready: (1) eosin (water soluble yellowish) 1:1000; (2) azure II, 1:1000. In making this mixture, 12 c.c. of the eosin solution and 9 c.c. of the azure solution are added to 100 c.c. of water.

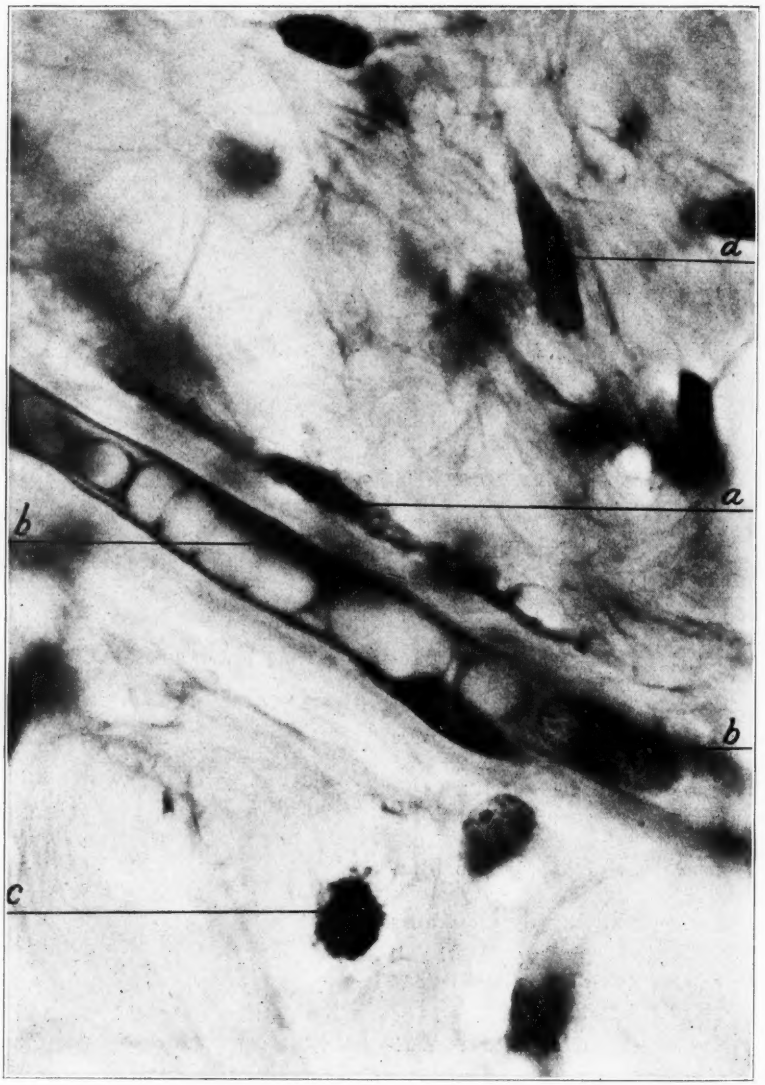

Fig. 2.—Normal human pulp. *a*, resting-wandering cell. *b*, endothelial cells. *c*, ameboid wandering cell. *d*, fibroblast. (× 1,500.)

ous fibers. The cells with which we are primarily concerned are the "resting-wandering cells" of Maximow, or histiocytes (*a*).

The nomenclature of these cells is not definite. Many investigators use their own terms. The first to see these cells was Metschnikoff, who termed them macrophages. Ranvier described them as clasmatocytes. Marchand calls them adventitial cells. Aschoff and Kiyono gave them the name histiocytes. In the following description, the term resting-wandering cells will be used.

Maximow describes them as developing in the period of embryonic life, from embryonic wandering cells which are hemocytoblasts (lymphocytes and myeloblasts). They wander in the loose connective tissue through the entire body, and when they cease their movement, they remain in the tissue as hemocytoblasts or become transformed into the resting-wandering cells. At the same time, embryonic fixed, but undifferentiated, connective-tissue cells may also be transformed into these cells. During the entire life of the individual, resting-wandering cells may develop from wandering lymphocytes. Peripheral vascular endothelial cells, as Maximow states, are not capable of being transformed into resting-wandering cells. He holds the same opinion concerning fibroblasts. This statement has been repeatedly questioned, especially recently by Möllendorff and his pupils. Maximow was not convinced by these investigators. These resting-wandering cells always retain their ability to move after certain forms of irritation, as, for example, in inflammation. They thus revert to their original conditions and again become wandering, ameboid cells.

They are recognized as phagocytes and storing cells. The latter expression means that if certain dyes are introduced into the living body, these cells will store the dye in their protoplasm. Through the use of dyes, the presence of these cells has already been observed in the dental pulp of animals. (Siegmund-Weber, W. Blotevogel, E. M. Goffung.) These properties differentiate them sharply from the fibroblasts, the common connective-tissue cells. Morphologically, they can also be distinguished in properly fixed and stained specimens of normal tissues. The nucleus of the resting-wandering cells is smaller and more deeply stained than that of the fibroblasts. This is clearly seen in Figure 2. The nucleus of the resting-wandering cell (*a*) is more flattened, ovoid and smaller than those of fibroblasts (*d*). In the nucleus of the fibroblasts, the chromatin is not so dense and dark, but is pale and dustlike, and one or more nucleoli are present. The protoplasm of the resting-wandering cells is darker than that of the fibroblasts, and is more granular in appearance. The cell outline is often notched, and several protoplasmic processes branch off. This is clearly seen in Figure 3.

The function of these cells should be evident from the description. In inflammatory reactions, they are transformed into ameboid phagocytes and play an important rôle in the defense reactions of the body. This is not their only function. It is claimed by investigators that these cells are those which produce the immune substances or antibodies in infections. Thus, those of a given area in the periphery may partake in so-called local immunity, while those of the hematopoietic organs take part in forming the generalized immune substances or properties.

In all three figures shown, up to now, we have seen the following cell elements

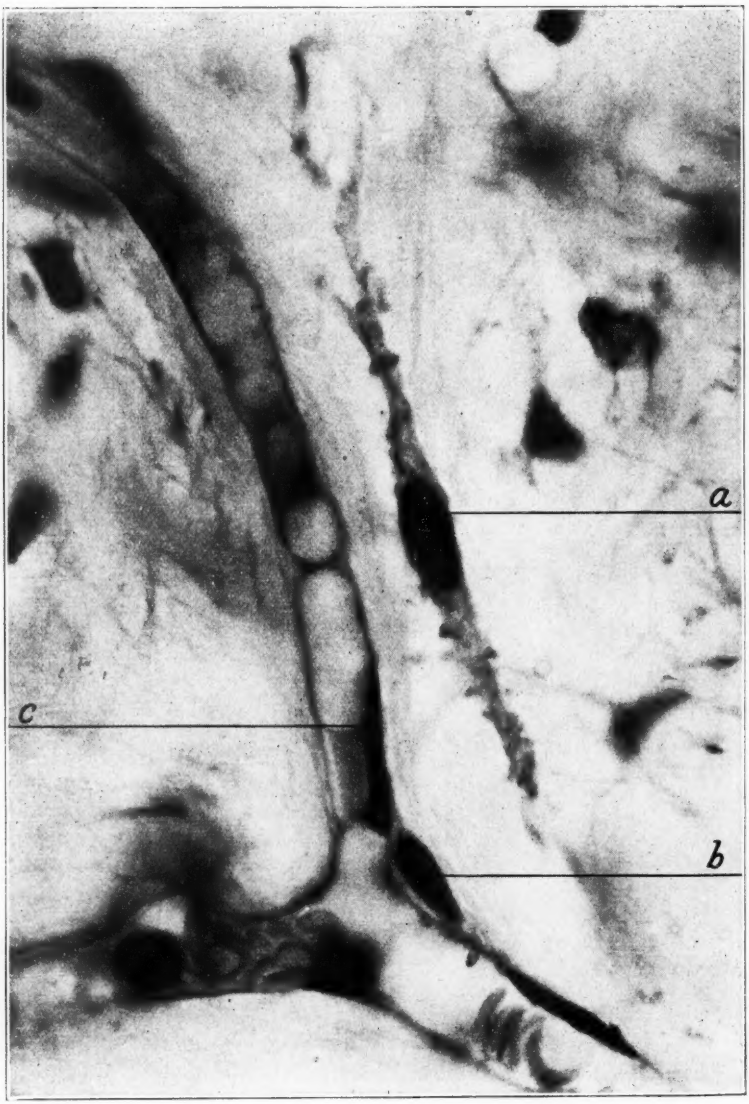

Fig. 3.—Normal human pulp. *a*, resting-wandering cell. *b*, undifferentiated mesenchyme cell. *c*, endothelial cell. (× 1,500.)

of the pulp: the fibroblasts, endothelial cells and resting-wandering cells.

A fourth important cell type, with which we are little acquainted, is the so-called undifferentiated perivascular mesenchyme cell, of Maximow. Some investigators call them pericytes. We see them in Figures 1 and 3, at *b*. They are very much like fibroblasts in appearance. As Maximow states, they are often not to be differentiated morphologically from common fibroblasts. Their nucleus is somewhat smaller; they have long protoplasmic processes, and they usually lie closely fitted to the outer wall of the small vessels. Maximow was the first to show clearly that these cells have the original properties of embryonic connective tissue cells. They are not differentiated in any way, and may become transformed into all the different types of connective-tissue and blood cells, if the adequate stimulus is present. In their normal state, they do not store dyes, but under the stimulus of inflammation they may become transformed into wandering cells as the resting-wandering cells do, and become storing phagocytic cells. They may also be transformed into fibroblasts or fat cells or, a special characteristic of their undifferentiated state, they may develop into cells producing the cellular elements of the blood.

In Figure 2, we observe the fifth cell type with which we are primarily concerned. At *c*, we see a round, somewhat notched cell with a relatively large kidney-shaped nucleus and a scanty protoplasmic body. This is an "ameboid wandering cell." Cells of this type vary in shape and size, the smallest being as large as a small lymphocyte found in the circulating blood. Three of these small wandering cells (*c*) are to be seen in Figure 4. In the same picture, we see a resting-wandering cell (*a*), undifferentiated mesenchyme cells (*b*), endothelial cells (*d*) and fibroblasts (*e*). The smaller of these wandering cells (*c*) most often have a round, indented nucleus. In the larger ones, the nucleus is kidney-shaped. The protoplasm is very limited in amount. They are not storing cells. Some of the smaller wandering cells are similar to the so-called monocytes. Maximow says that these wandering cells not only resemble lymphocytes and monocytes, but also are identical with them. This does not mean that all of them have migrated out of the vessels: the undifferentiated mesenchyme cells also are capable of producing lymphocytoid or monocytoid wandering cells. The latter wandering cells may never have been within and may never enter into the blood stream, and are therefore called histogenous wandering cells. The greater number of ameboid wandering cells in normal tissues originate from the lymphocytes or monocytes of the blood stream. These are called hematogenous wandering cells. Morphologically, there are no differences between the two types.

It is perhaps well to explain the differences between some of the white blood elements, such as leukocytes, lymphocytes and monocytes. There are granular and nongranular leukocytes. The granular type is what we more commonly call leukocytes. They may contain basophilic or eosinophilic granules, but generally contain neutrophilic granules. The nongranular leukocytes are the lymphocytes and monocytes. Maximow considers the lymphocytes the primitive free connective-tissue cell from which all connective tissues develop. The most extensive morphologic and experimental study of the lymphocytes and monocytes was made by William Bloom. He showed conclusively that the monocytes develop from the lymphocytes, and pointed out

Fig. 4.—Normal human pulp. *a*, resting-wandering cell. *b*, undifferentiated mesenchyme cells. *c*, ameboid wandering cells. *d*, endothelial cell. *e*, fibroblast. (× 1,500.)

that it is often impossible to differentiate some of them morphologically. It is important to know that the terms monocyte and macrophage do not mean the same and must not be confused. The macrophages are large cells of the connective tissue which have arisen from fixed resting-wandering cells and from the nongranular leukocytes of the blood; while the monocytes are small cells of the blood stream which have developed from lymphocytes, and which normally enter the connective tissue in but small numbers.

Returning to the ameboid wandering cells: Maximow has shown that they play an important part in inflammatory reactions. After the lymphocytes and monocytes migrate from the vessels, owing to inflammatory irritation, they grow rapidly and become phagocytic cells. Maximow termed them polyblasts, indicating by the name their varied appearance, their multiple function and their wide developmental potentialities. The term polyblast includes not only the cells which develop from migrated lymphocytes or monocytes, but also those which develop from fixed connective-tissue cells; namely, the undifferentiated mesenchyme cells, and more particularly the resting-wandering cells. As stated by Maximow, under an inflammatory stimulus, the resting-wandering cells become wandering cells: they "wake up," mobilize, become active and are transformed into large, ameboid, phagocytic cells. These are the histogenous polyblasts. Hematogenous polyblasts, which develop from the migrating lymphocytes and monocytes, cannot be distinguished in later stages of inflammation from histogenous polyblasts.

Figure 5 is a photomicrograph of the normal human pulp showing the tissue around a small vein. A resting-wandering cell with a peculiarly shaped body is seen at *a*. Between this cell and the wall of the vessel, we see an undifferentiated mesenchyme cell (*d*), and at *b*, an ameboid wandering cell. Inside the blood vessel, red blood corpuscles and lymphocytes (*c*) or monocytes can be seen. The fibrous layer bordering the vessel is composed of collagenous fibers.

The presence of these different cells in the human pulp is not surprising. But in the study of the normal histology, they have not received proper consideration. It is certain that in the future we must study the biologic as well as the pathologic changes in the pulp by concentrating our interest on the function and changes in these cells.

In the periodontal tissues, the same tissue elements can be found. In several dental articles and books of recent years, we read about the wandering, storing and phagocytic cells, but no clear explanation of their true nature has been given. We find the most information in the book of Siegmund and Weber, "The Histopathology of the Oral Cavity," and in the book by Häupl and Lang, called "Paradentitis Marginalis" (their term for the disease commonly called pyorrhea). Siegmund himself conducted many investigations in the field of inflammation in general, and is an authority on the subject. Lang, who treats the question in his book written in cooperation with Häupl, is a pupil of Maximow, having spent some months in his laboratory. These investigators touch the subject, but do not enter into explanations. Nor do they picture the different cells, but merely describe their presence.

In Figures 6-22, we present pictures of human specimens, both normal and pathologic. Figure 6 is a resting-wandering cell from the loose connective tissue of the periodontal membrane of an adult human being. It is known that

Fig. 5.—Normal human pulp. *a*, resting-wandering cell. *b*, ameboid wandering cell. *c*, lymphocyte in the blood vessel. *d*, undifferentiated mesenchyme cell. (× 1,500.)

between the principal collagenous fibers of the periodontal membrane, there are areas of soft, loose connective tissue which surround the blood vessels, nerve fibers, and lymphatics. In this tissue, we find all the elements of connective tissue which were found in the pulp and which are described as characteristic of loose connective tissue in general. Resting-wandering cells, ameboid wandering cells and undifferentiated mesenchyme cells are present. These cells can be found not only in the periodontal membrane but also in the surrounding marrow spaces of the alveolar bone. They are particularly numerous in the bone channels through which the blood vessels and nerves enter the pulp canal.

In addition to the cells mentioned above, we find a great number of connective tissue mast cells. In Figure 7, we observe one of these mast cells in the company of a resting-wandering cell and a fibroblast. This picture offers a good opportunity for comparing the fibroblast with the resting-wandering cell. The difference in the three types of nuclei is quite striking. The mast cell contains a small round nucleus partially obscured by the many basophilic granules in the protoplasm. We have not seen these mast cells in the normal pulp. The bone channel just mentioned contains them in great abundance. Their function is not known, although it seems that they have some kind of secretory function. Some authors claim that they develop from granular leukocytes or plasma cells, although they probably originate from lymphocytes.

In the case of a chronic abscess at the apex of an upper first bicuspid, as shown in Figures 8-22, we have the opportunity to study the different types of cells concerned in the inflammatory reaction. Maximow describes three different cell types involved in such a reaction:

1. The fibroblasts or common connective-tissue cells, whose function it is to produce a fibrous capsule to isolate the injurious agent from the surrounding tissues. The fibroblasts proliferate by mitosis and replace the tissue destroyed by the inflammation, thus forming the scar.

2. The neutrophil granular leukocytes normally found only in the blood stream, which are the first new cell type to appear in the field of inflammation. Their function is generally believed to be to digest and destroy the invading bacteria. They are the special phagocytes termed microphages by Metschnikoff. They not only phagocytose bacteria but also produce different substances somewhat of the nature of a ferment, which act on the bacterial agents causing the inflammation. These substances supposedly neutralize and detoxify the injurious material; but only when the leukocytes are destroyed are these substances liberated. These leukocytes which appear first in inflammation degenerate and are destroyed very rapidly, and in the later stages of inflammation are not present.

3. The "polyblasts" of Maximow. The development of this type of cell has been discussed in the paragraphs dealing with the dental pulp. As was stated, Maximow claims that they have a double origin: they arise partly from fixed cells (histogenous polyblasts) and partly from nongranular blood leukocytes (hematogenous polyblasts.) The local fixed cells which develop into polyblasts are the resting-wandering cells. Under the influence of the inflammatory stimulus, they "wake up," become mobilized and are transformed into large ameboid phagocytes.

In this case, we can observe the formation of the polyblasts and their ultimate

Fig. 6.—Resting-wandering cell in the normal periodontal membrane of a human being. (× 2,000.)

fate. Figure 8 is a section of an abscess at the apex of the first bicuspid. The apex of the second bicuspid also reaches into the abscess area. A higher magnification (Fig. 9) shows the fibrous capsule around the abscess, isolating it from the surrounding tissues. The difference in the tissue outside and inside the fibrous capsule is clearly evident. A still higher magnification is shown in Figure 10, in which the fibrous bundles between the inflammatory cells can be seen. Further magnifications of different areas outside and inside the capsule show the transformation of the different cells into polyblasts, and their function.

Figure 11 is a picture of an area outside the fibrous capsule of the abscess. A resting-wandering cell (*a*), a mast cell (*c*) and a lymphocyte (*b*) (an ameboid wandering cell) can be seen in this picture. The cell with the large, jagged, round protoplasmic body, and round, dark nucleus (*d*) is a histogenous polyblast which developed from a resting-wandering cell. A fibroblast (*e*) is seen in the field with a pale and distinctly different nucleus.

Figure 12 is another photomicrograph of a region outside the fibrous abscess capsule. Here, a resting-wandering cell (*a*) is to be observed with a body somewhat differently shaped from those seen before in Figures 6, 7 or 11. The body is not so long and slender as before, the protoplasmic processes are rounding off and the nucleus is becoming round. This we may assume to be a resting-wandering cell in the process of transformation into an ameboid histogenous polyblast.

In Figure 13, cell *a* is farther along in its transformation into a polyblast, showing a rounding off of its protoplasmic body and presenting a kidney-shaped nucleus.

Maximow could not demonstrate the development of histogenous polyblasts from fibroblasts or endothelial cells. Many investigators believe that the fibroblasts or the endothelial cells are the main source of the polyblasts, but Maximow believes that the endothelial cells are highly differentiated cells, not having any of the properties of the undifferentiated mesenchymal elements. The only developmental possibility for the endothelial cells which has actually been observed is their transformation into common fibroblasts, which are considered by Maximow to be cells which have ordinarily reached the limit of their development, excepting their transformation into osteoblasts or ostoclasts. The function of the fibroblasts in the defense reaction is to produce the intercellular substance for the development of the collagenous fibers which wall off the inflamed area.

The blood cells which are transformed into polyblasts in the field of inflammation are the lymphocytes and monocytes which migrate from the blood vessels at the same time as the granular leukocytes. The latter degenerate and disappear very soon, but the migration of the lymphocytes continues. After migration, the lymphocytes and monocytes enlarge to such an extent that, after a certain time, they cannot be distinguished from the polyblasts which have developed from resting-wandering cells.

Figure 14 shows an area in which a differentiation can still be made. Two histogenous polyblasts (*a*) and a hematogenous polyblast (*b*) may be seen. In the same picture, a plasma cell may also be seen at *c*. Special attention should be given to the difference in nuclei of these cells. The plasma cells, which develop from lymphocytes, are a type of polyblast. They are found also in the normal tissues, especially in active glands and

lymph nodes, seldom in loose connective tissue. In chronic inflammations (especially in certain types, such as syphilis, Hodgkin's disease and tuberculosis), they are present in great abundance. The protoplasm is large and stains dark at the periphery. The eccentric nucleus is relatively small. The arrangement of the chromatin is characteristic, having the appearance of the spokes in a wheel. The plasma cells have not the power of further development. After a time, they degenerate and disappear. As a sign of their degeneration, we find acidophil

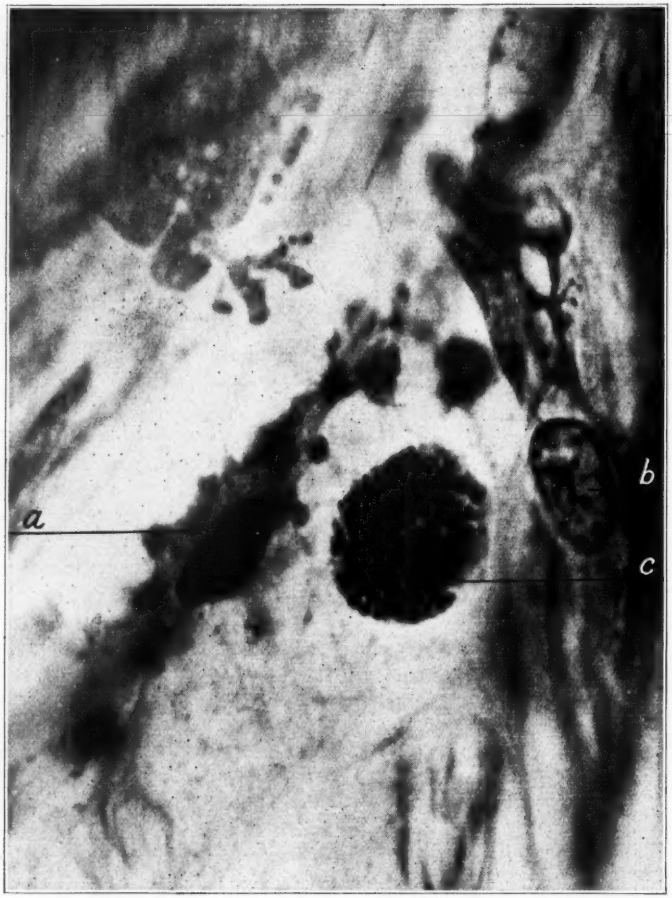

Fig. 7.—Resting-wandering cell (*a*), fibroblast (*b*) and connective tissue mast cell (*c*) from a bone channel through which the vessels and nerves enter the apex of the tooth. (× 2,000.)

granules, the so-called Russell's bodies in the plasma cells. These bodies enlarge and become free in the tissue. The function of the plasma cells may possibly be to elaborate some secretion or to produce certain antibodies. Some investigators believe that they have an important function in digesting decomposed protein material.

Inside the abscess wall, we can hardly differentiate between the various types of polyblasts. We see only small and large round cells with round or irregular nuclei, as shown in Figure 15. Here, two large polyblasts are present at *a*, closely resembling plasma cells. A large polyblast (macrophage) is seen at *b*. In the protoplasm of this cell, we see vacuoles which in life may have been fat droplets taken up as the result of phagocytosis or storing. Lymphocytes (*c*) are to be seen scattered in the tissue. The lymphocyte at *d* is developing a larger protoplasmic body. This is a step in the transformation into a polyblast. In Figure 16 at *b*, a similar transformation may be seen. At *a* in the same picture, we observe a large polyblast with vacuoles in its protoplasm. It is not possible to determine from which source this polyblast developed—the histiocytes or the lymphocytes. The fibroblasts at *c* are easily differentiated by their pale nuclei and ill-defined protoplasmic bodies.

Figure 17 shows the tissue around a blood vessel. Polyblasts of different sizes and shapes are seen scattered throughout the field. At *a*, we observe typical plasma cells, and at *b*, polyblasts with round or kidney-shaped nuclei. The cell at *c* may also be considered a polyblast, probably of histogenous origin; or it may be that this cell is in the process of a transformation from a polyblast back into a resting-wandering cell. As has been stated before, Maximow asserts that the polyblasts, after fulfilling their functions in the field of inflammation, may revert back to resting-wandering cells or become transformed into common fibroblasts.

On the interior of this abscess, different fields disclose widely differing conditions. In one area, degeneration and phagocytosis of cells can be observed, which shows the activity of the polyblasts. In other areas, the inflammatory activity seems to be abating, and scar tissue is being formed. Figure 18 illustrates the former. Lymphocytes (*a*), large polyblasts, some of the plasma cell type (*b*), a few fibroblasts (*c*) and a scanty intercellular substance are present. Figure 19 shows even more activity. Dead cell elements may be seen engulfed in the protoplasm of the polyblasts, as at *a*. The exact nature of the structure at *b* cannot be determined, as it is not possible to follow it in a series of sections, because of the great abundance of cells around it. It is perhaps a polyblast which has engulfed another smaller polyblast, or it may be a small blood vessel with a polyblast in its lumen.

In Figure 20 is shown another area containing dead cell elements scattered throughout the field, some of them free in the tissues (*a*) and some engulfed in the polyblasts (*b*). Some of the polyblasts in this figure have a peculiar appearance (*c*). The cytoplasm of these cells has been almost entirely replaced by huge vacuoles surrounding the nuclei. As previously mentioned, these vacuoles were most probably filled with fat during life. This fat probably represents the end-product of destroyed and engulfed cells. Sometimes this storing of fat gives rise to a foamlike appearance of the cytoplasm. Siegmund and Weber observed these "foam cells" in dental

granulomas, and advanced the theory that they were fat-storing histiocytes.

In the last two photomicrographs (Figs. 21-22), the beginning of the resolution of the abscess is shown by the transformation of the polyblasts into resting-wandering cells or into fibroblasts. At *a*, Figure 21, we see cells that greatly resemble

Fig. 8.—Abscess on the apex of an upper bicuspid of a human being. (× 13.)

resting-wandering cells, although they differ very little from fibroblasts in the

field. Their nuclei are as yet darker and smaller than those of the fibroblasts (*b*). This area may be considered as representing scar formation in its inception. Polyblasts (*c*) small and large are seen scattered throughout the field, but not in large numbers as in those areas showing great phagocytic activity (Figs. 18-20). Instead, we find many more fibroblasts and cells closely resembling them.

In Figure 22, further resolution is shown by the presence of a larger amount of fibrous matrix. Many fibroblasts (*a*) are present, but the intercellular substance is more abundant than are the cells. At *b* is seen a cell nucleus which is being destroyed and disintegrated; polyblasts small (*c*) and large (*d*) and cells (*e*) resembling the plasma cells are also present. The whole picture shows the prevalence of a fibrous intercellular substance, the basis of scar tissue.

This scar formation probably is the end-result of the fight between the tissues and the bacteria. The battle in this case seems to end in the victory of the tissues. Bacteria and dead cells are engulfed and digested. Toxins and other products of the bacterial invasion seem to be neutralized. In one area, the battle goes on; in the other, repair work starts. The destroyed tissues are replaced by connective-tissue cells and fibers. Why in some cases the bacteria and in others the organisms win out, I shall not discuss in this paper. I should like only to quote a paragraph from one of Maximow's papers:

> The so-called constitution, the endogenous individual peculiarities of an organism which manifest themselves under both physiologic and pathologic circumstances and especially in the course of inflammatory processes and of general infections, seems also to depend mostly on the qualities of the "mesenchyme."

CONCLUSIONS

1. With the proper histologic technic, the different cell types in normal as well as pathologic conditions can be easily recognized and differentiated.

2. In the dental pulp, we find the same cell elements that have been recognized and differentiated by Maximow in the loose connective tissues elsewhere in the body.

3. The same cells are also present in the tissues surrounding the roots of the teeth.

4. Inflammatory reactions occur in the periodontal tissues in the same way as elsewhere in the body. The result of an inflammatory reaction will depend on the exogenous and endogenous factors: the virulence of the bacteria and the response of the reacting mesenchyme.

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Fig. 9.—Higher magnification of the abscess shown in Figure 8. The difference between the tissues outside and those inside the abscess capsule is evident. (× 60.)

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Fig. 10.—Higher magnification of the abscess wall. Collagenous bundles build the capsule. (× 280.)

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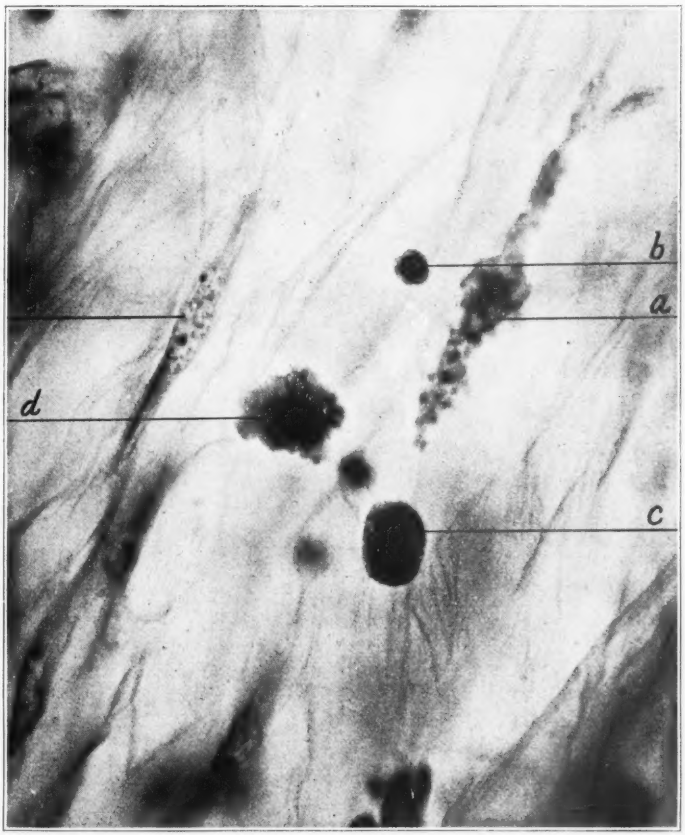

Fig. 11.—Cellular elements from outside the abscess capsule. *a*, resting-wandering cell. *b*, ameboid wandering cell (lymphocyte). *c*, connective tissue mast cell. *d*, polyblast (histogenous). *e*, fibroblast. (× 1,300.)

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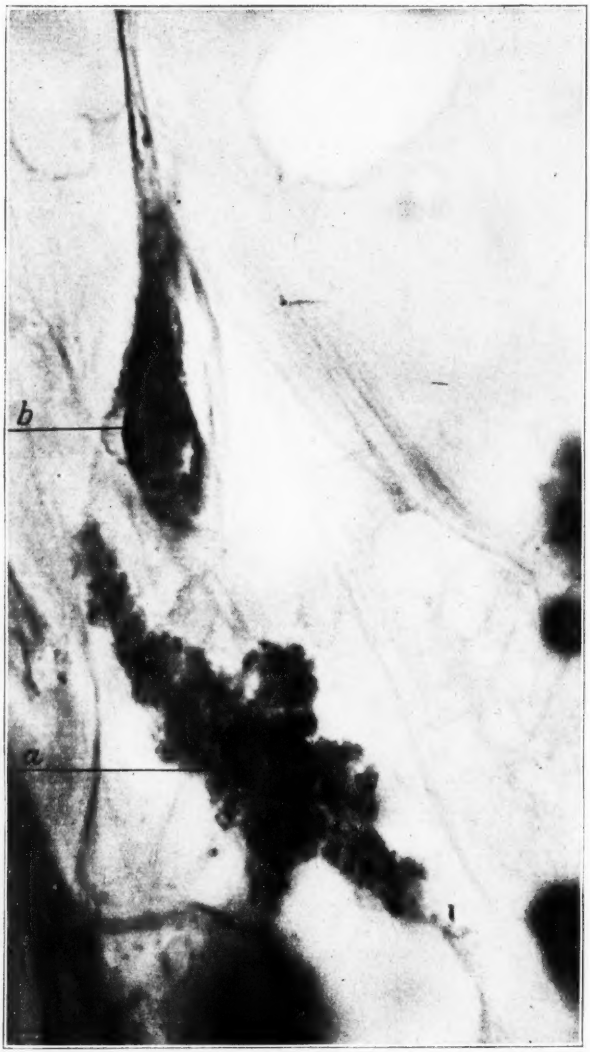

Fig. 12.—*a*, resting-wandering cell. *b*, fibroblast. The resting-wandering cell (*a*) becomes transformed into a polyblast owing to the inflammatory stimulus. The protoplasmic processes are rounding off and the nucleus is becoming round. (× 2,000.)

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Fig. 13.—Rounding off of the resting-wandering cells (*a*) near the abscess capsule. *b*, fibroblast. (× 2,000.)

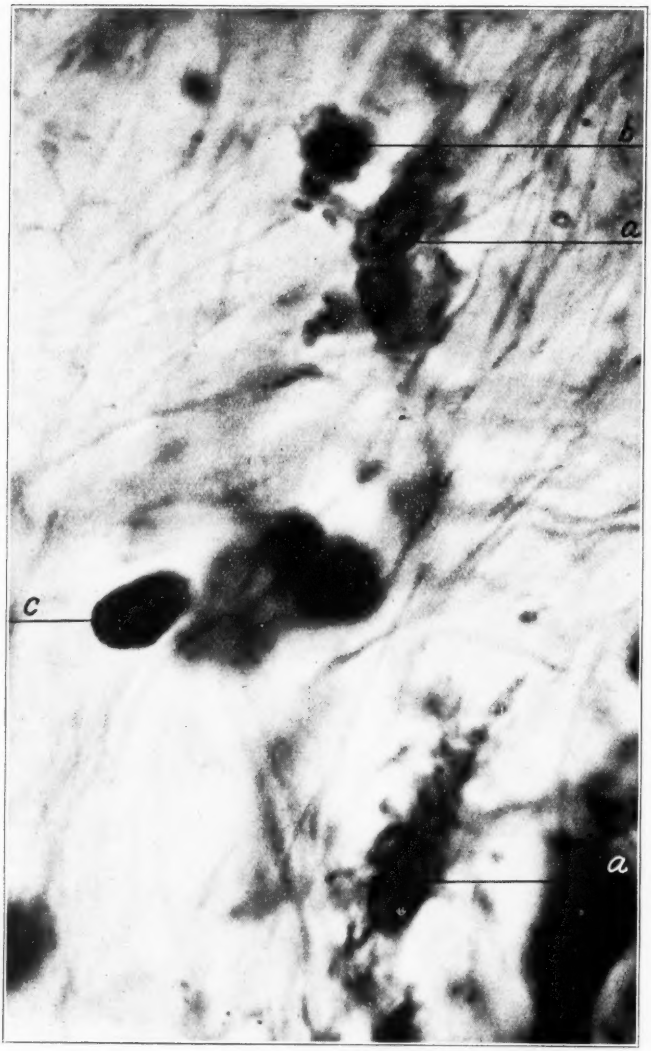

Fig. 14.—Histogenous (*a*) and hematogenous (*b*) polyblasts. *c*, plasma cell. (× 2,000.)

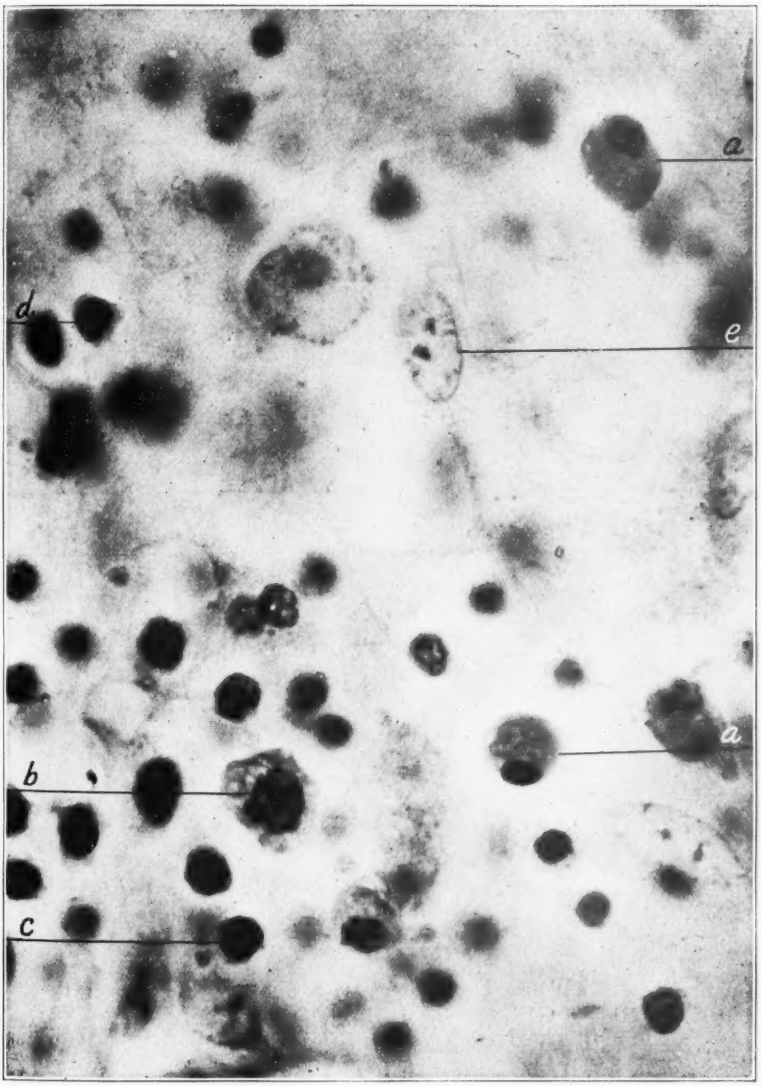

Fig. 15.—Polyblasts of different shapes and sizes in the abscess. *a*, polyblasts closely resembling plasma cells. *b*, large polyblasts with vacuoles in the cytoplasm. *c*, lymphocyte. *d*, lymphocyte with somewhat larger protoplasmic body. *e*, fibroblast. (× 1,800.)

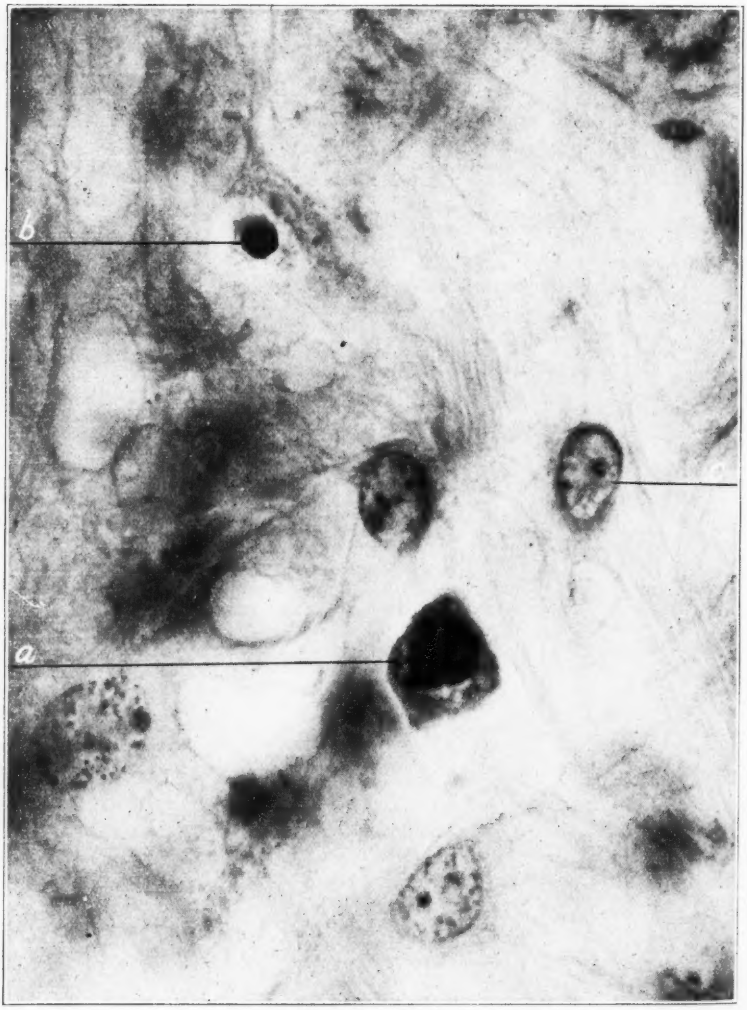

Fig. 16.—Large polyblast (*a*) with vacuoles in the cytoplasm. *b*, lymphocyte in process of becoming a polyblast. The protoplasm is larger and irregularly outlined. *c*, fibroblast. (× 1,800.)

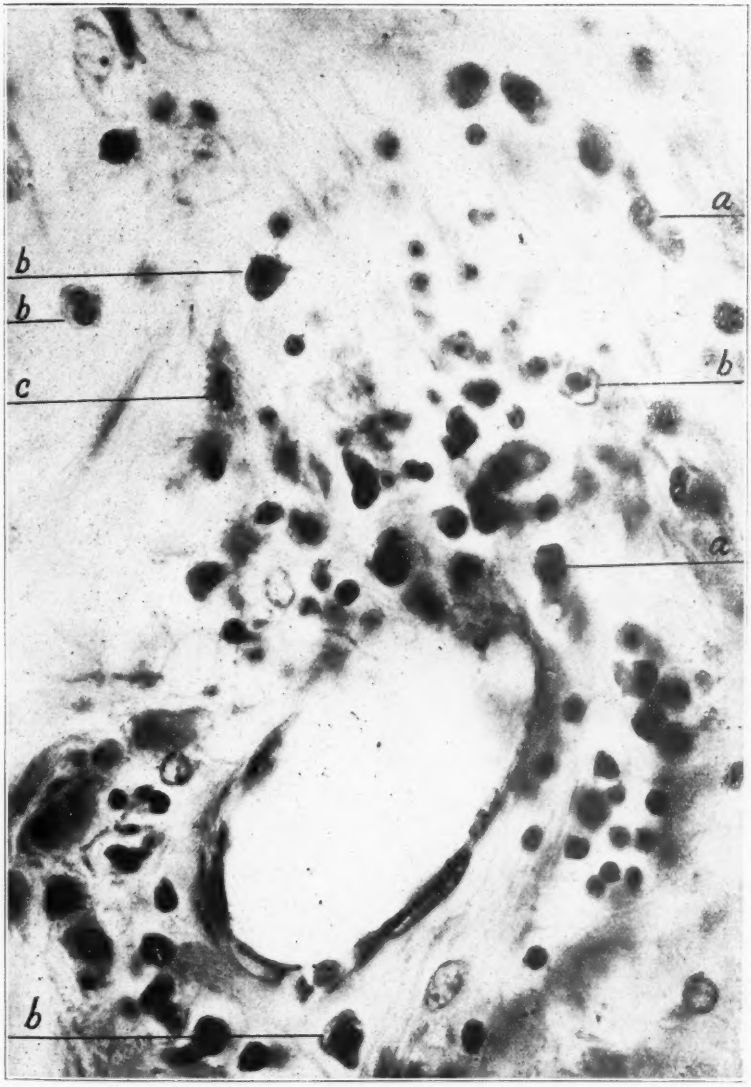

Fig. 17.—Polyblasts around a blood vessel in the abscess. *a*, plasma cells. *b*, polyblasts of different sizes and shapes. *c*, polyblast probably of histogenous origin or in process of transformation into a resting-wandering cell. (× 1,050.)

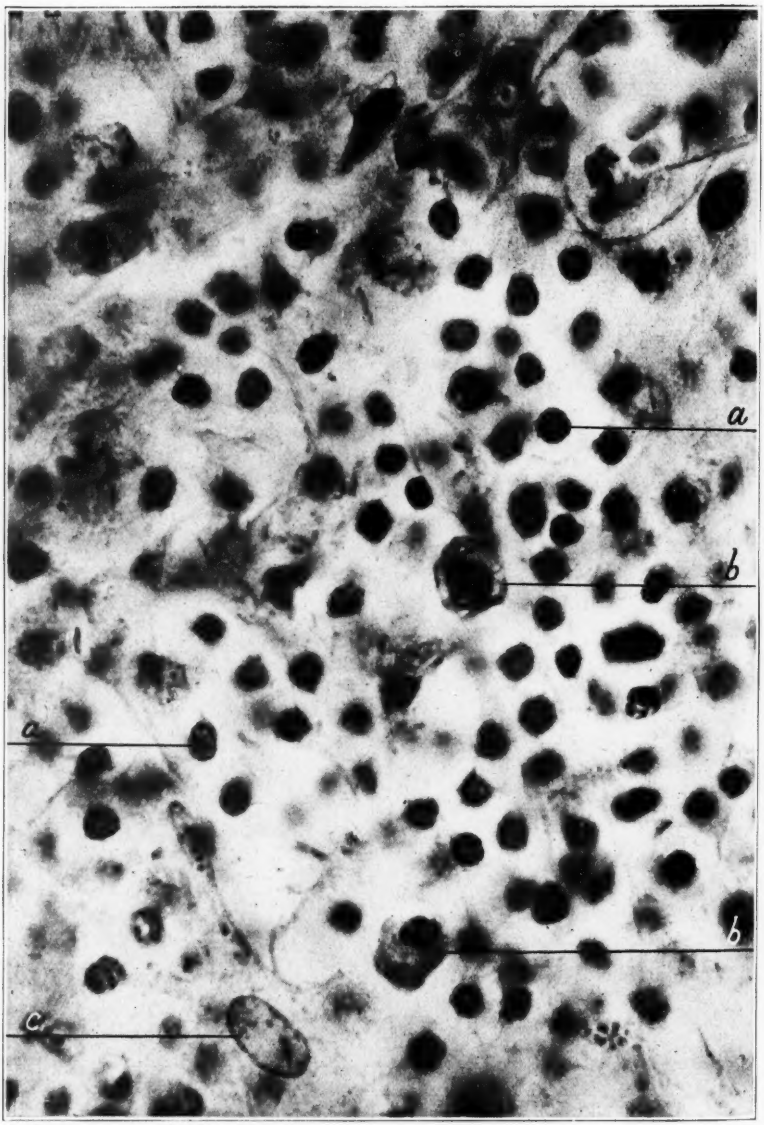

Fig. 18.—Area in the abscess. Wandering cells of different sizes. *a*, small lymphocytes. *b*, large polyblasts with vacuoles. *c*, fibroblasts. (× 1,400.)

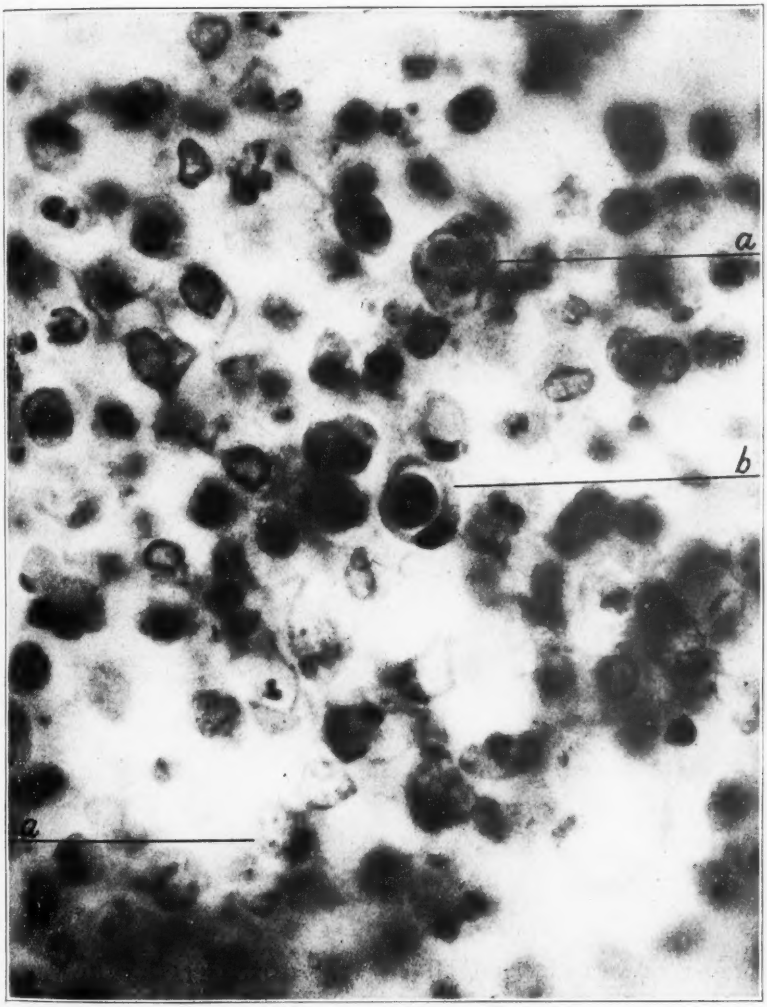

Fig. 19.—Polyblasts (*a*) engulfing the remnants of destroyed cells. *b*, a phagocyte with a smaller polyblast in its protoplasm, or probably a small blood vessel with the cell in its lumen. (× 1,400.)

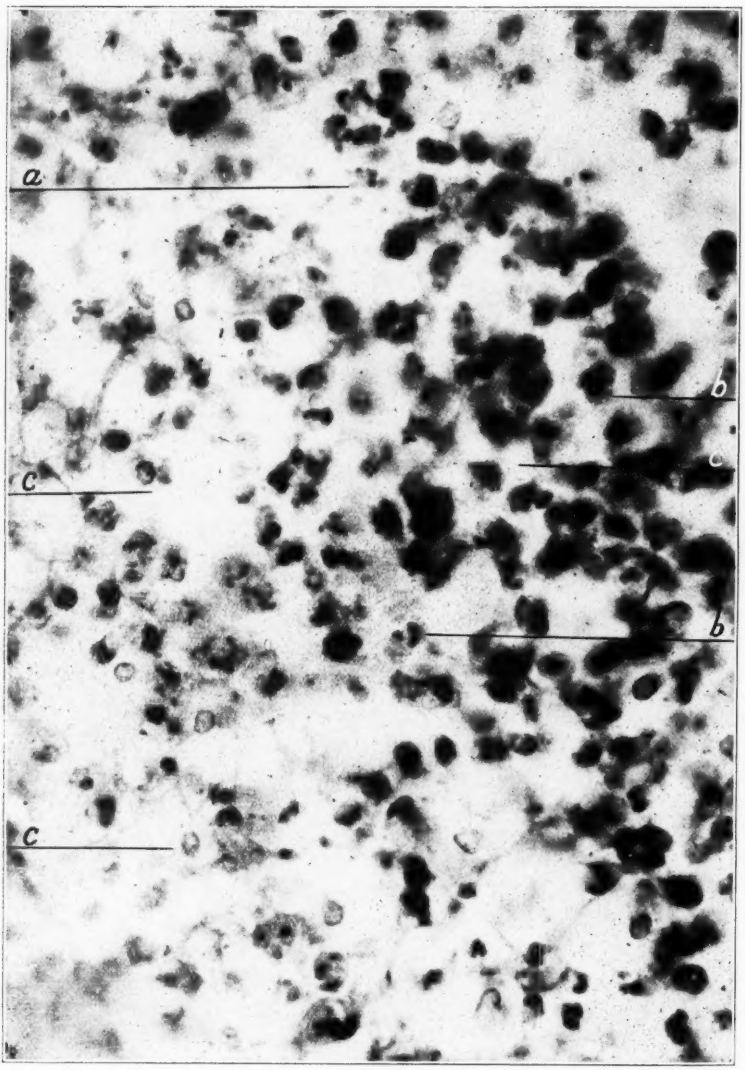

Fig. 20.—Polyblasts actively engulfing the remnants of destroyed cells. *a*, cell remnants free in the tissue. *b*, polyblasts with enclosures. *c*, polyblasts with stored fat in their cytoplasm. (× 820.)

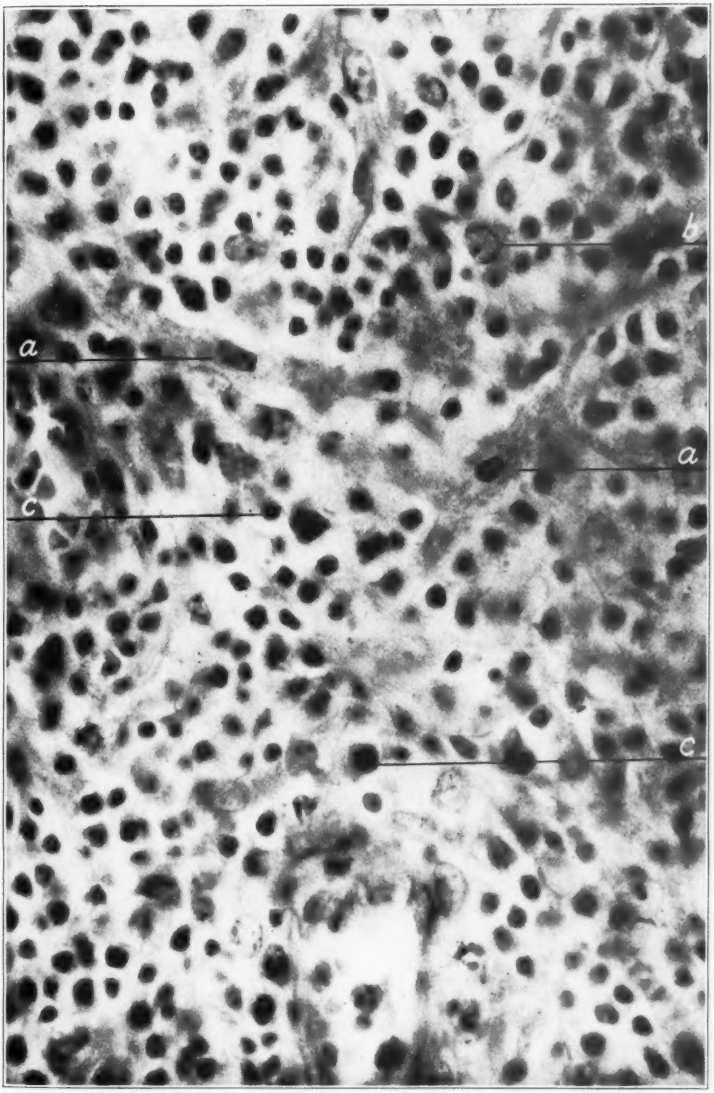

Fig. 21.—*a*, polyblasts which probably become resting-wandering cells. *b*, fibroblasts. *c*, polyblasts. The fibroblasts (*b*) are more numerous in this area (inside the abscess capsule). (× 820.)

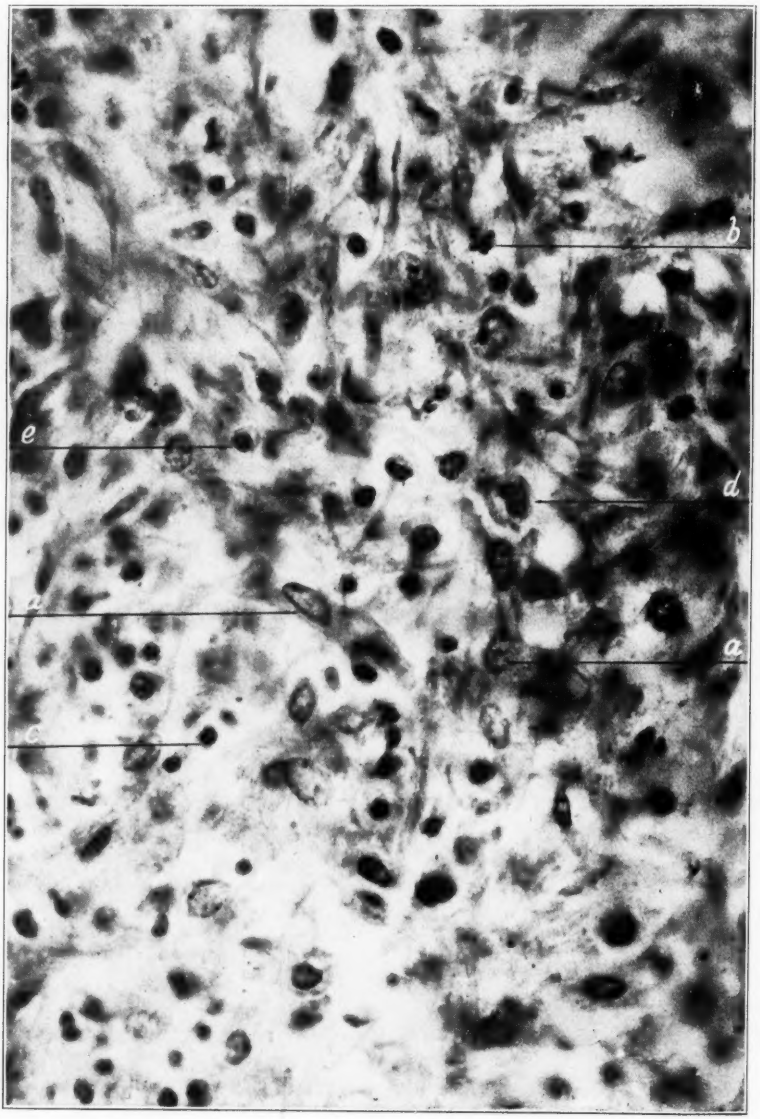

Fig. 22.—Fibroblasts (*a*) and a fibrous intercellular substance outweighing the wandering cells. *b*, destroyed cell nucleus. *c*, small polyblast. *d*, larger polyblast. *e*, plasma cell. (× 820.)

MOUTH INFECTIONS IN RELATION TO GENERAL HEALTH*

By ISAAC A. ABT, M.D., Chicago, Ill.

IT is with a great deal of temerity and with a great degree of humility that I appear before this learned society tonight to discuss the subject with which the members of this organization are intimately acquainted and of which I am only a casual observer. It seems pedantic to enter into a discussion of the relative importance of organs in the animal body; whether it be in health or disease, all organs are important. It is fundamentally necessary to good health that all should be functioning normally. The mouth and its adnexa have many important functions in the preservation of health, and in quite the same degree present possible relationships to morbid processes too numerous to mention.

We have not only to consider the diseases which occur in the mucosa of the mouth, gums and tongue and the diseases of the teeth and salivary glands, but we must also bear in mind that the oral cavity is probably the most important atrium for organisms to enter the digestive tract as well as the respiratory and circulatory systems. We are not surprised to find that some of these bacteria gain lodgment on the abrasions in the mouth and produce local manifestations of disease. But it is not so simple as one might at first suppose. While the mucosa of the mouth is a most perfect recipient for the growth and development of micro-organisms, these minute parasites show a decided preference or a predilection for certain tissues and organs. The pneumococcus prefers the lung; the diphtheria organism thrives best in the pharynx, on the tonsil and in the larynx, and the organism producing thrush thrives best on the mucosa of the mouth. Vincent's spirillum shows a preference for the mouth and pharynx; and, as is so well known, the oral cavity harbors a great number of organisms in health. The mouth of the new-born baby is sterile. It is generally thought that bacterial growth begins about the sixth hour after birth. When the baby is 9 or 10 hours old, bacteria may be obtained by culture from the mucous surfaces. *Streptococcus salivarius* occupies an important place, being constantly present during the first few years.

After the teeth have appeared, the flora of the mouth changes and, in addition to *Streptococcus salivarius,* such anaerobes as lepthorix, various spirochetes, spirilla and other organisms are found. *Endameba buccalis* is frequently found in mouths that are badly cared for or when the teeth are carious. No pathologic importance is ascribed to it. It is noteworthy that when the mouth is well cared for, the saliva is neutral or slightly alkaline, and the flora aerobic or facultatively anaerobic; while in mouths that are badly cared for, the saliva is

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strongly acid and the flora anaerobic with putrefaction.

The eruption of the teeth and their qualitative condition is an important index of health and development during infancy and childhood. It is a well recognized fact that, in rachitis, the eruption of the teeth is delayed and the quality of their structure, after eruption, is materially influenced by early rachitic processes.

When the calcium level of the blood and the calcium metabolism of the infantile organism are diminished, a condition is produced which is variously spoken of as spasmophilia or tetany. Some years ago, I collected a group of children who gave a history of repeated convulsions due to tetany during the nursing period and who, in later years, showed the residual effects of an associated rickets. By reason of the data obtained, it was assumed that these infants had suffered from manifest tetany during infancy. The permanent teeth showed enamel erosions, probably as the result of a calcium deficiency during their evolution. I have seen several of these children with lamellar cataracts associated with tetany.

There can be no doubt that the condition of the teeth must depend in part at least on the general health of the child. Malnutrition and rickets in early infancy are important factors in tooth development and indeed in the stability of the permanent teeth. The condition which is called caries in the adult may depend on ill-health or improper nourishment or, to state it another way, on the occurrence of rickets and spasmophilia during infantile life. The modern study of vitamins, proper feeding and hygiene during young life must have the greatest interest to those practitioners who are called on to treat abnormalities of dentition in older childhood and adult life.

The effect of congenital syphilis on temporary as well as on permanent teeth need only be mentioned in passing, as this condition is so well known to every student of stomatology.

The relation of the thyroid gland to the development of the osseous system is well recognized. In a child suffering from cretinism, the development of the bony skeleton is much delayed. A 4 months old child may have the bony development of a new born infant. The fontanel may remain open unusually long, even for twenty years. All of the facial bones are undeveloped, and thus we explain the saddle nose, the small eyes and the prominent forehead of the cretin. The jaw bones are likewise gracile; the milk teeth appear late and may persist. The permanent teeth are small and abnormally located, erupt late and decay early. The permanent teeth are frequently widely and irregularly spaced and deficient in enamel. Some time ago, a marked cretin came to me who presented all the earmarks of severe hypothyroidism. The disease had been unrecognized and the child hitherto untreated. No teeth had erupted. I was astonished to find that, after several months of vigorous thyroid treatment, the child not only improved, but began to grow in length, and the teeth erupted in rapid succession.

I have digressed from the main topic in order that I might point out the relation of the oral cavity to the entire organism. As specialists, we attempt to separate one group of organs from another, and we are too prone to believe that we are dealing with a localized condition, whether it be disease, such as infection or disturbed function, or normal growth or development. The fact is

generally recognized (it hardly deserves comment) that no single organ can be considered of and by itself alone. It is a part of the whole, and whatever affects the growth and development of any particular structure will affect in some degree the organism as a whole.

Wherever there is infection or localized disease, whether it involves the tongue or the mucosa of the mouth or a tooth, the entire organism may be more or less affected.

The oral mucous membrane of the young child may be the seat of diseased conditions, some of them local infectious processes, while others are secondary to constitutional disturbances or disease in other parts of the body. Mercury, bismuth, phosphorus or other drugs may produce serious mouth manifestations. In infancy and young childhood, the mucous membrane of the cavity of the mouth is no less delicate than other parts of the body and consequently abrasions followed by infections are relatively frequent. These infections were more common during that period when every baby's mouth was cleansed by swabbing it out with antiseptic solutions at rather short intervals. In the same way, the pacifier has produced erosions of the mucosa. While mouth cleansing was almost universally practiced formerly, it has in the last decade become almost obsolete. Lesions of varying severity were produced, from catarrhal to septic stomatitis. In some cases, ulcerative stomatitis followed.

Mouth lesions are of great significance in the infant's development. Even the mildest forms interfere with the ingestion of food on account of painful sensations in the mouth. Some of the severer oral infections in infancy are associated with long continued fever. Occasionally symptoms of a general infection become manifest, and hemorrhagic nephritis may occur. Many severe gastro-intestinal disturbances, with diarrhea, loss of weight and vomiting, are associated with various forms of stomatitis. Finkelstein studied statistically the effect of stomatitis on the mortality of infants admitted to his hospital. He showed that out of 100 children admitted to his institution, those suffering from stomatitis during the first three months of their lives showed a mortality of 24 per cent. The stomatitis, though not the cause of death, bore some causal relationship to fatal disease which ensued. Controlling this group, he observed another series who had healthy mouths and who were of the same age as the previous group. In the latter, he found a mortality of only 8.5 per cent.

Finkelstein wisely observes that the mouth infections of the present day are of greater significance than the umbilical infections. In former times, infantile sepsis was a very frequent disease. This condition has largely disappeared with the advance of aseptic treatment of the cord.

In some obstetrical institutions where mouth washing is still in vogue, veritable epidemics of oral infections occur.

An interesting case in point is the occurrence of thrush, which, as is well known, is caused by a yeast or fungus organism, *Oidium albicans,* or *Sacchromyces albicans.* The lesion consists of a whitish or yellowish, sometimes brownish, deposit, usually resembling milk curds, which is slightly raised from the red mucous membrane and is adherent to it. It is localized on the tongue, mucous membrane of the cheeks or the tonsil. Sometimes, the lesion becomes confluent and forms large plaques. The organism which produces thrush is nearly always present in the mouth of normal young infants. Under ordinary circum-

stances, conditions do not exist which favor the growth of this organism. If pathologic changes occur in the gastro-intestinal tract, the yeastlike organism finds a favorable soil and proceeds to produce the characteristic lesion. If the gastro-intestinal disturbance persists, the extent of the lesion progresses and soon covers more or less of the mucous membrane of the mouth, the esophagus and even the stomach or intestines. If the gastro-intestinal lesion improves or is cured, the thrush disappears of its own accord. In young infants in whom the mouth has been cleansed and abrasions produced, this morbid condition may also occur. The outstanding fact is that those infants who are suffering from intestinal disorders are unusually predisposed to infection with thrush. This illustrates the manner in which the mouth reflects pathologic processes in remote organs.

I recently saw a necropsy on a 4 months old baby that was breast fed and had had repeated diarrhea, vomiting and loss of weight, with thrush infection of the mouth since its fourth week. Necropsy revealed thrush in the mouth as well as in the esophagus, throughout its entire extent.

A very common and annoying condition that receives scant attention in the textbooks or current literature is *fetor ex ore,* sometimes alluded to as halitosis. This condition, which may be due to a variety of causes, will be briefly referred to as a subject worthy of consideration. Discussion may be permitted on account of its practical importance.

It is generally assumed that *fetor ex ore* arises from decomposition processes about the teeth or is due to gastric or intestinal conditions. There can be no doubt that acute indigestions, febrile infections and chronic gastro-intestinal disturbances, as well as the diseases of the esophagus, may arise after pathologic processes in the mouth cavity.

The question that interests us most is what part the mouth cavity plays in the development of *fetor ex ore.* It is certain that carious teeth or inflammatory processes in and about these appendages may lead to an intensely disagreeable odor from the mouth. It is also well recognized that plugs in the crypts of the tonsils may lead to fetor. The same is true of suppurative processes in the accessory sinuses. In the majority of these cases, the infection is due to streptococci, though this infection may be associated with staphylococci, pneumococci and other organisms. In addition to the mouth cavity itself, the tongue may harbor decomposition products, but not every coated tongue gives off an offensive odor. One finds putrefaction occurring, particularly when the patient himself complains about unpleasant and bad taste in the mouth, usually associated with poor appetite. Even in these cases, the tongue in its entirety does not participate in the decomposition process. More particularly, the root of the tongue with its circumvallate papillae is involved. In the same region may be found the mucous albuminous and follicular glands. These occur in great numbers and proceed backwards to the epiglottis. The follicular glands at the base of the tongue constitute the lingual tonsil. The secretion from these crypts is exuded on the root of the tongue. The secretion may become obstructed in the ducts and bacteria may collect and develop in them. The remains of food may also gain access to these follicles, and decomposition may occur and a bad odor result. It may be easily demonstrated by introducing a spatula into the mouth, and scraping off the secretion from the root of the

tongue or pressing out some of the secretion from the ducts and glands, when a foul smelling substance is obtained. If one examines this material under the microscope, it is discovered that bacteria and fungi in great numbers are contained in it. This foul odor from the base of the tongue occurs particularly when there is indigestion accompanied by vomiting. The vomited material remains attached to the root of the tongue or some of it enters the ducts. In acute febrile infections, when the tongue is partly dry or the food is improper or consists largely of milk, accumulations take place in this region. In the latter cases, the fetor subsides as soon as the symptoms and fever have abated.

There are persons who suffer from chronic *fetor ex lingua,* which may persist for a long time and eventually produce glossitis. This infection at the root of the tongue may be the focus for infection of the gastro-intestinal tract and possibly also the biliary system; or the infection may find its way into the circulation and lead to a variety of metastatic processes.

I. Boas[1] says that, in the course of years, he has observed cases of chronic intestinal disturbances characterized by partial regurgitation, tympany and diarrhea, associated with *fetor ex ore,* which were improved by systematic cleansing of the root of the tongue. He also observed numerous cases of dyspepsia due to infection in the lingual tonsil.

On the other hand, putrefaction at the root of the tongue may be due to diseases of the stomach or intestine. It should be remembered also that glossitis may occur as a result of disease of the teeth, tonsils and accessory sinuses. Consequently, in diagnosing *fetor ex lingua,* the latter conditions must be excluded. In numerous cases, too, a patient may suffer from *fetor ex lingua* and the gastro-intestinal tract remain entirely normal. It must not be overlooked that the fetor may be due entirely to gastro-intestinal or hepatic causes; or there may be disturbances in the esophagus with putrefactive changes going on in this tube. Carcinoma in the upper third, or diverticula, may produce a foul smell from the mouth. One should differentiate between genuine fetor from the mouth and fetid regurgitation. The latter occurs in stagnating processes of the stomach, such as pyloric stenosis; or in carcinoma, the eructation of gas may contain hydrogen sulphid and the odor permeate the entire room.

It should be observed in passing that the mouth is not very frequently infected from the gastro-intestinal tract. Boas has observed the discharge of putrid pus from the stomach in patients suffering from carcinoma giving rise to a foul odor without infecting the mouth.

It has been suggested by certain investigators that putrifying foods producing foul smelling gas might be absorbed from the stomach or the large intestine into the general circulation and eliminated through the mouth. This seems rather far fetched and cannot be accepted without skepticism.

Recently, Eppinger and Umber have spoken of a fetor of hepatic origin. Eppinger calls attention to a peculiar indol-like odor detected in the breath and the mouth, and stresses the importance of this particular smell in the diagnosis of grave icterus. Umber observed this odor, maintaining that it occurs in acute yellow atrophy and in cirrhosis of the liver. He describes it as a peculiar, sweet, aromatic odor that is exhaled from the mouth and may fill the entire room.

1. Boas, I.: Therap. der Gegenwart, January, 1929.

Boyce has also observed this *fetor hepaticus,* particularly in severe cases of catarrhal jaundice. The patients complain also of a bitter taste in the mouth that must be associated with the absorption of bile stuffs. A fetid odor from the mouth may arise also from atrophic rhinitis or antrum disease; stomatitis, whether due to infection or a nutritional disorder such as scurvy, or to chemical changes produced by bismuth or mercury or phosphorus or lead; negligence in cleaning the teeth, or the ingestion of certain foods, for example, garlic and onions, or certain drugs such as cacodylates. There is no odor more difficult to bear than that which arises from noma, which is so frequently associated with ulcerative stomatitis. The sickening and almost unendurable stench which is exhaled by patients suffering from pulmonary gangrene, fetid bronchitis and bronchiectasis need only be mentioned in passing. A urinous odor to the breath resembling that of trimethylamin is observed in uremia.

The subject of Vincent's infection is so thoroughly familiar to all who are interested in diseases of the mouth that it seems superfluous to enter into a detailed discussion. This disease is caused by the fusiform bacillus associated with Vincent's spirillum. It is thought that the spirillum is a degenerative form of the bacillus. The disease is characterized by the production of a membrane which, when removed, reveals an ulcerated surface tending to bleed readily. In some instances, the infection produces no symptoms and is discovered only by accident, though the disease may be associated with ulcerative stomatitis and gingivitis.

There is usually an enlargement of the cervical glands on the affected side. In uncomplicated cases of Vincent's infection or stomatitis, the process remains almost entirely local. The constitutional symptoms which accompany other forms of mouth and throat infection are either slight or absent, though some severe types of this disease have been reported.

Redlich[2] recently reported the case of a child of 3 years in whom a membrane was present in the mouth and on the tonsil. The child suffered from a severe anemia, was unable to take food and became pale and emaciated. The cervical glands became markedly enlarged; the spleen was readily palpated, and the red blood cells were reduced in number (hemoglobin, 38). The child improved after the administration of neo-arsphenamin. The outstanding symptom was marked anemia and morphologic changes in the character of the erythrocytes and lymphocytes.

Another interesting condition which has been recently described and which has attracted wide attention on the part of physicians and dentists is the so-called agranulocytic angina.[3]

In 1922, W. Schultz called attention to a group of cases showing severe gangrenous stomatitis and an unusual blood picture, occurring mostly in women of middle age who had previously been well. The onset of the disease was usually sudden and was associated with fever, chills and malaise. One or both tonsils and the pharynx became injected and necrotic. The lymph nodes in the region of the affected parts were enlarged. Jaundice was present, with no bleeding from the skin. The liver and spleen were frequently enlarged. On examination of the blood, the red cells, hemoglobin and platelets were normal. In the white blood cells, the character-

2. Redlich, F.: Ztschr. f. Kinderh., 45, 361, 1928.

3. Kastlin, G. J.: Am. J. M. S., 173:799-813 (June) 1927.

istic changes were present. They were greatly reduced in number and, on the differential count, the polymorphonuclear leukocytes were decreased or absent. In most instances, the lymphocytes constituted about 100 per cent of the leukocyte field. In Schultz's series, all the patients died in three to four days of pneumonia. At necropsy, the bone marrow was intensely red. On microscopic examination, it was poor in cells, with almost complete absence of granulocytic types. Because of the peculiar damage to the granulocytic blood cells associated with stomatitis, Schultz attached to the condition the name agranulocytic angina.

In considering the report of Schultz, there were those who thought that the disease was a clinical entity which had been previously undescribed. Others thought that leukemia and certain forms of sepsis might produce similar blood changes, with stomatitis. As the reports of this disease continued to come in, it was found that it varied in severity as well as in symptomatology. Very soon, a patient was presented who, contrary to Schultz's original prognosis, recovered. While Schultz found the disease only in females, the case of one male patient was subsequently reported.

Clinical description of the condition of the mouth may be interesting at this point.[3] A woman, aged 24, showed marked *fetor ex ore,* as well as discoloration and hemorrhage about the gum margin of two teeth. The tongue was slightly coated but moist. There was an area of ulceration on the mucous membrane of the mouth opposite the last right molar tooth. The throat was inflamed. The tonsils were not enlarged. The patient had moderate fever and tachycardia. The blood examination showed: red blood cells normal in number; hemoglobin reduced, and white blood cells numbering 2,500 per cubic millimeter the lymphocytes constituting 74 per cent of the entire count. A few days later, the temperature fluctuated between 101 and 105. The white blood cells fell progressively to 1,100 and finally to 500 cells per cubic millimeter, and after eight days in the hospital, the patient died.

Up to June, 1927, there were forty-three cases reported of which thirty-four occurred females. One case in a child 4½ years old is reported. The oldest individual was 74. The average age of the females was 46 years, and of the males, 29 years.

Ulcers have been observed in various parts of the body, hemorrhages into the skin and into the viscera (discovered at necropsy). Out of the forty-three patients reported, forty died and three recovered. One patient reported by Friedman was recovering from the gangrene of the mouth, and the condition of the blood as ascertained by microscopic examination also improved; but the patient contracted pneumonia and died. As has already been remarked, the symptoms vary, though, in every case reported, stomatitis has been present. The disease bears some resemblance in its clinical manifestations to leukemia and also to aleukemic leukemia, though severe sepsis may produce similar symptoms. Two observers have found *Bacillus pyocyaneus* and have conjectured that this organism might be specific in the production of agranulocytic angina. This cannot be confirmed. Acute poisoning may produce a clinical course not dissimilar to agranulocytic angina. Thus, thorium, arsenic, benzene and the roentgen rays may produce such a lesion. This disease is of great interest in such a discussion as this, because the oral manifestation may be but a part of a severe general disturbance. The fever and profound blood changes,

with frequent occurrence of a terminal pneumonia, are characteristic of the disease.

Another condition similar to agranulocytosis is infectious mononucleosis. This condition, which occurs in children and adults during grippal infections, is characterized by glandular enlargement, with marked lymphocytosis, the large lymphocytes usually being the predominant ones. These patients recover, as a rule, in contradistinction to those suffering from agranulocytic angina.

Acute leukemia, aleukemia and aplastic anemia may produce ulcerating necrotic processes in the mouth and pharynx, with hemorrhages and ulcer formation on the gums and in the mouth.

Foot and mouth disease is a very contagious affection of domestic animals, especially cattle and hogs, and is communicable to man. It is characterized by the appearance of a vesicular eruption in the mouth and pharynx and on the skin, especially in the interdigital spaces. It may be difficult to differentiate this affection in man from the common aphthous stomatitis, particularly if one confines his attention only to the mouth cavity. The affection spreads to the nose and to the skin in the region of the mouth, though the skin over the breasts and sternum, as well as the cutaneous covering of the neck, chin, arms and legs and especially the interdigital parts, may be involved. Fever and marked prostration are usually associated with the disease.

Human beings coming directly in contact with diseased cattle or drinking milk or consuming milk products from infected goats and cows may contract the disease. In England, 205 persons contracted the disorder during the course of one week. A case is reported of twins who acquired the disease from the ingestion of goat's milk. One died after several days illness; the other recovered. The incubation period varies from two to fourteen days.

When children are attacked, they usually suffer from gastro-intestinal catarrh, loss of appetite and a general feeling of depression. Vesicles varying in size from a pinhead to a pea appear on the mucous membranes of the mouth. These rupture in a few days and leave ulcers, which tend to persist and are usually accompanied by profuse salivation. Swallowing is difficult and the increased flow of saliva makes it imperative to empty the mouth frequently. Vomiting and diarrhea occur often in children. In addition, the disease is characterized by pains in the extremities, headaches, dizziness and more or less prostration. In man, the disease usually runs a mild course, though in young and weakened children, complications are likely to occur. Ordinarily, the disease runs its course in from three to six weeks.

One might continue in great detail and consider at great length a number of constitutional diseases and infections associated with mouth lesions. One may briefly enumerate a few of these. It is said that ulcers may occur on the gums in dysentery. It is a well known fact that the pustules of variola as well as the vesicles of varicella may appear in the mouth early in the disease. In septicemia, pustules and ulcers are not infrequently observed. Gonorrheal infection of the mouth manifesting itself as an ulcerative, membranous stomatitis has been observed, usually characterized by the production of a pseudomembrane and purulent secretion, with erosions on the mucous membrane of the gums and of the buccal mucosa.

Typhoid ulcers may occur over the soft palate. They vary in size from a

pinhead to a pea. They usually present themselves as white raised spots. Such ulcers may also occur in pneumonia and pleurisy. The pneumococcus may produce ulcers on the tongue, palate and the inner aspects of the cheek.

Tuberculous ulcers may be observed on the mucous membrane of the pharynx, tonsils and tongue. They usually are quite painful and are often associated with a severe pulmonary or laryngeal tuberculosis. They are irregular in shape, circumscribed and covered by a pseudomembranous exudate, and, in the periphery, one may find small gray, tuberculous nodules. From these ulcers, the tubercle bacillus may be recovered.

One is tempted to refer to the focal infections, but would hesitate. So much has been written on the subject, both pro and con, that the literature is replete and the printed page groans under the burden.

The clinical and experimental evidence so far adduced convinces us that, in certain instances, active infections in the mouth about the teeth, as well as tonsillar infections, may produce disease in remote organs, or may produce systemic infections. On the other hand, there can be no doubt that this hypothesis has met its limitations, and oral and dental infections are not guilty of all the ills with which they have been charged.

Dental decay is extremely prevalent in childhood, and its immediate effects interfere with the ingestion and mastication of food. A child who swallows his food whole because of painful or decaying teeth is almost certain to suffer from disturbances of digestion and possibly other defects of constitution. Dental caries is also associated with apical abscesses and gingivitis, and, occasionally, stomatitis. That these local infections may further interfere with the ingestion of food, produce systemic effects and cause anemia, fever, loss of appetite and headache cannot be disputed by any clinician of experience. Dental infection is often said to be associated in a causal way with rheumatism and heart disease. Just how frequently this source of infection occurs in young life is a mooted question. To quote one author, Howe: Out of 50,000 children examined, 40,000 suffered from dental infections and not one of them had arthritis or endocarditis. In a study of a group of rheumatic children, it has been observed that the incidence of dental caries was not perceptibly greater than in nonrheumatic children. This agrees with the studies of Poynton and his coworkers, who are not convinced that dental infection played a rôle in rheumatic diseases of childhood. The outstanding fact is that the dental caries of childhood should be prevented whenever possible and treated as early in life as is feasible to prevent the dental infections of later life.

It is interesting to note the controversial position among eminent investigators about "oral sepsis." On the one hand, we have the diligent researches of Rosenow, who believes that dental disorder may be a source of infection to the entire organism. For example, he maintains that streptococci from dental infections may cause rheumatism, myositis, anemia, myocarditis, pericarditis, ulcers of the stomach, cholecystitis, pancreatitis, appendicitis, nephritis, iritis, thyroiditis, herpes zoster, chorea, sciatica, neuritis, parotitis epidemica, erythema nodosum and nephrolithiasis.

William Hunter, in a classic paper on the rôle of sepsis and antisepsis in medicine, originated the modern usage of the term "oral sepsis" in 1900. He says that this matter of oral sepsis is of urgent importance in relation to the whole multifarious and widespread group of infec-

tions, medical, surgical and dental, caused by the actual presence or toxic action of pyogenic organisms. Hunter believed that he had discovered the key to ill-health, and that it was the mission of the oral surgeons and dentists to unlock the gates of the new era of health by their magic touch.

Schottmüller, a German investigator, among others, has reviewed the work of Rosenow and his adherents and of William Hunter. He objects to the expression "oral sepsis" introduced by Hunter. He thinks that the designation is not a happy one. He says, first, that the diseases mentioned by Hunter as sequelae, such as anemia, gastritis, purpura, nephritis and rheumatism, cannot be considered as septic conditions but should be thought of as metastatic inflammatory processes in other organs.

By "sepsis," it is understood that a condition exists in which a septic focus has established itself and that from this source bacteria or their demonstrable products are either continuously or intermittently being poured into the blood or lymph streams. The author referred to maintains that neither pathologic or bacteriologic studies of the blood and tissues show that a condition of real sepsis exists. He does maintain that these infections of the dental system are local in character and that they may become the atria for pathogenic organisms and thus produce systemic or metastatic processes. He concludes that, as a matter of pathologic and clinical experience, purulent metastases in remote parts of the body arising from dental infection are very rare.

Schottmüller must not be misunderstood. He does not take the extreme position of those who would deny the importance or existence of dental infection as a cause of disease in other organs.

It has ever been so in medical practice: when an innovation is introduced, the pendulum has taken on additional speed and swung exceedingly far, and some time has elapsed before the vibrations resumed their normal periodicity.

Most of us think now that the early enthusiasts regarding dental infections were overzealous, not only in their beliefs but also in their treatment. The best minds in dentistry and medicine are now following the swing of the pendulum back to a more conservative position.

In this brief review, I have attempted to summarize some of the salient points in reference to infections of the mouth and the concomitant constitutional manifestations. I have avoided, for the most part, a discussion of controversial subjects, as well as those which have purely academic as opposed to practical interest. I have attempted to show that the diseases of the mouth may play an important part in causing not only local but also constitutional symptoms. I have also attempted to call attention again to the fact that a careful examination of the mouth may, in many instances, reveal the health status of the patient and may be a valuable index of disease threatened or in progress.

FUNDAMENTAL PRINCIPLES IN THE PREPARATION OF THE MOUTH FOR BRIDGEWORK*

By LEE WALTER DOXTATER, D.D.S., New York City

IN the past, the approach to the problem of bridgework in both practice and theoretical consideration has been what might be called historical, rather than scientific. In the history of bridgework, it is readily seen that the first thought of the prosthetist was to provide a structure which would meet the mechanical and esthetic demands. In other words, the early prosthetist was faced with the necessity of building an appliance which would be strong enough to withstand the muscular effort made by the patient and also be retained in position. The matter of appearance was always and is now an important consideration, but was secondary to the factors mentioned. The question of comfort and of oral health at first received but little attention, but as mechanical improvements were made, these factors were recognized as being of paramount importance. Until recent times, the earlier trend of thought continued to prevail; and, even today, it is safe to say that most prosthetists think first of the purely physical requirements of the case.

Under these circumstances, I feel that my first message should deal with the biologic factor in bridgework. In other words, I wish to stress the need for studying each case requiring bridgework from the standpoint of the state of health of the mouth at the time work is begun and also from the standpoint of the probable effect of the restoration on the health of the mouth. I furthermore wish to emphasize the point suggested in the title of this paper, namely, the preparation of the mouth for bridgework, a thing which must precede, but which is nevertheless an integral part of the management of the case.

In approaching this problem, we should first consider what effect bridgework may have on the oral tissues. The most conspicious injury which has been traced to bridgework is trauma to the periodontal tissues. So conspicious has this been that there was a time when it was considered almost inevitable that teeth which were used as bridge abutments would develop intractable periodontal disease. This problem has received much attention from prosthetists, and great progress has been made toward its solution. Another form of injury associated with bridgework, although not so directly to be charged to it, is periapical disease. This problem also has been attacked with considerable success.

The question of both periodontal and periapical disease must be studied as the fundamental and primary step in bridge construction, and only after the case has been studied from this standpoint should the dentist begin to plan the type of restoration which he is to put

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in the mouth. If this premise is granted, it becomes logical to approach the problem frankly from the standpoint of the periodontist. Stillman and McCall have consistently, over a period of years, called attention to the state of the occlusion as the fundamental factor in periodontal health. Their studies on traumatic occlusion have led them to insist on the establishment of what is usually called balanced occlusion as the primary requisite in securing periodontal health. In treating cases of periodontal disease, they attempt to establish an occlusal condition throughout the mouth which will provide for the most satisfactory working of the dental mechanism. In doing this, they frequently remodel teeth which may not in themselves be affected seriously by periodontoclasia. The occlusal condition thus obtained I have preferred to call functional occlusion, a term given to the dental profession by Ottolengui. This term is used because of the fact that balanced occlusion, as it is understood by the prosthetist, is not always attainable in the natural dentition, whereas, a partially balanced occlusion, adequate for health, may usually be developed.

Functional occlusion has been defined by Ottolengui as "such arrangement of the teeth as will provide the highest efficiency during all the movements of the mandible necessary to the function of mastication." In certain cases, it is found that a full functional occlusion cannot be obtained without undue sacrifice of tooth structure, and in these cases, a partial functional occlusion is to be sought. This has been defined by me as "that arrangement of the occluding surfaces which will bring the greatest practicable number of teeth into harmonious contact in the various movements of the mandible, without harmful sacrifice of tooth structure." If the establishment of functional occlusion either full or partial is regarded as necessary in the treatment of periodontal disease, it is obvious that it is equally important in prevention. I therefore propose the hypothesis that the mouth in which bridgework is to be placed should be given the highest degree of functional occlusion obtainable before beginning the construction of any piece of bridgework. This statement holds true even when the mouth exhibits no perceptible symptoms of periodontal disease when the case is undertaken.

In studying the mouth from this standpoint, we are concerned first with the general form and relationship of the dental arches. When previous extraction has resulted in drifting or elongation of teeth, the first attention is given to the reduction of prominent teeth or cusps to the general occlusal level found in the mouth. This may call for the grinding down of teeth which are in supraclusion, or the building up by means of inlays or other restorations of teeth which are below the line of occlusion. We next consider the occlusion of the anterior teeth as to the vertical overbite and also as to the balance of these teeth when in protrusive occlusion. Excessive vertical overbite usually indicates a disharmony as between the incisal guidance, the posterior cusp guidance and the condyle inclination. This disharmony invariably means an abnormally heavy stress on the incisors in all protrusive movements and should be relieved to as great an extent as is possible. In these cases, the leverage on these teeth in the protrusive occlusion is greater than in the normal case and may be the source of a severe periodontal lesion. In the protrusive occlusion, it is even more important that a balance be

obtained which will distribute stress evenly over the largest practicable number of teeth. By this one expedient alone, much disease may be avoided.

Another occlusal requirement which has been noted by Stillman and McCall as being important in the preservation of health and also in the treatment of disease is the elimination of close interlocking of the cusps of the posterior teeth. A common example of this and one readily observed is the close overlapping of the buccal cusps of the upper bicuspids and molars over the buccal cusps of the lower teeth. This close overlapping is also frequently seen in the cuspid region, where it is usually emphasized by an exaggerated vertical overbite. When this condition is found, it should be corrected by modification of the offending cusps. The ideal here is the establishment of such relations of the teeth as will permit free lateral movements of the mandible with a ready gliding of the lower teeth along the cusp inclines of the upper teeth.

Only when the highest obtainable functional occlusion has been secured is the prosthetist ready to undertake the active planning and construction of the case. No mention has been made as yet of the study of periapical conditions, but it is to be understood that this subject has already been given the fullest consideration. Teeth which show palpable periapical lesions are by that fact barred from consideration as bridge abutments or even for retention in a mouth in which a bridge is to be placed. The latter part of this statement may be questioned, but it must be remembered that the prosthetist of today can look farther ahead as to the probable events in his patient's mouth than could the prosthetist of yesterday. He may not only predict with considerable success the trend of things in any given mouth, but he is also able to place bridgework with greater confidence that it will give long and valuable service. He should therefore plan for that mouth a general program which is not likely to be upset by future extractions. A bridge may be properly considered a failure if it is found necessary to reconstruct it within a short time because of the extraction of teeth elsewhere in the mouth. Such eventualities should be and may usually be foreseen.

To return to the subject of periodontal disease: The next step is the treatment of such disease if it is found anywhere in the mouth. It is, of course, a fact that the complete treatment of such disease may not be accomplished in all cases until contact points have been established and drifting of teeth has been arrested by the insertion of restorations. Treatment should be carried as far as possible previous to the construction of the bridgework.

In planning the restoration of missing teeth, the dentist of today has before him the choice of a wide variety of types of bridges and methods of retention. His choice among these should not be governed by the enthusiasm of the advocate of any one type of work or by personal predilection for any technic in which he may have gained special proficiency. Almost any type of bridgework or attachment now being advocated by specialists in this field may be used in properly selected cases. Fixed bridgework has its place and removable bridgework is of course frequently indicated. Perhaps the most unfortunate feature in modern bridgework is the tendency to place removable bridges in any mouth in which there are a sufficient number of teeth remaining to serve for their mechanical retention. Many cases in which

bridgework might conceivably be placed would be better served by the insertion of partial dentures. In many cases also, the best interests of the patient indicate the extraction of remaining teeth and the construction of full dentures.

Referring to an earlier statement that the dentist may now, in most cases, foresee the developments of the future: It is evident that all cases involving the restoration of a considerable number of teeth must be studied with regard to whether they are likely in the future to become full denture cases. In mouths which show a high susceptibility to periodontal disease and in which it is probable that the home care by the patient will be inadequate, the question arises as to whether the progress of this disease may result in the more or less complete loss of the alveolar ridges. If this seems likely to occur, the best policy is to extract the remaining teeth at once, in order that the final ridge form may be as favorable as possible to the construction of successful dentures. This will, of course, call for advice, which the patient may be unwilling to accept; but the duty of the dentist is nevertheless clear. Other cases calling for similar advice are those in which the construction of suitable bridge abutments may endanger the vitality of the pulps or may actually call for devitalization. If the health of the patient is below par or if the patient has already developed recognizable symptoms of metastatic disease, the need for extraction and the placing of full dentures is even more definitely indicated.

This situation may be more graphically presented by the description of a hypothetical case. If, for instance, the patient has the following teeth in the mouth: right upper third molar, right second bicuspid and lateral incisor, left central incisor, the left upper cuspid and second bicuspid, and it is found that these teeth have already been attacked by caries to such an extent that devitalization would be required for the construction of attachments for removable bridgework, it is usually the part of wisdom to advise a partial denture. The reason for this is that such teeth must usually be deeply cut into and often banded for the purpose of utilizing internal attachments. When this is done, the effect of gingival irritation, together with the excessive leverage imposed by the appliance, is such as to lead to the early development of serious pathologic conditions. Furthermore, it should be noted that the anterior teeth are never suited to the application of internal attachments, with the possible exception of devitalized cuspids. A better procedure is to place porcelain jacket crowns on any anterior teeth that have been deeply attacked by caries and three-quarter crowns on any posterior teeth that have been weakened by caries. A partial denture with paralleled wrought gold wire clasps is then constructed. If the economic factor contraindicates this plan, the carious anterior teeth may be restored with three-quarter crowns, and well-executed amalgam fillings may be inserted in the posterior teeth. A partial denture is then constructed. In any event, the use of so many bands in one case, whatever the type of appliance, is to be condemned.

In general, it may be said that the important thing is not so much the type of appliance which is finally agreed on as the fact that the dentist shall recognize the ultimate needs of the case and shall so advise his patient. His position in this respect is like that of the surgeon, who must recognize the need for certain operations but may be unable to

prevail on his patient to have them performed. His chief responsibility is to make a correct diagnosis and to give the proper advice.

If more teeth remain in the mouth, the insertion of bridges rather than partial dentures may usually be recommended. In general, if the span or spans to be filled are short and the abutment teeth have adequate alveolar support, fixed bridgework is usually indicated. The choice of attachment will vary with the location in the mouth and the condition of the abutment teeth. In many cases, the three-quarter crown will be the attachment of choice. This is especially true when the teeth show a decided tendency to approximal or lingual decay, as this crown provides greater protection for the teeth than does the inlay. In many cases, even if the tooth has already been attacked by caries, the mesioclusal or distoclusal inlay will provide adequate retention. If a mesioclusodistal inlay is required, it is my usual procedure to make this inlay in sections, placing first that part of the inlay which is not to act as the bridge support and providing in this an occlusal lock for anchorage for that part of the inlay which is to act as the bridge attachment. Correct cavity preparation will provide adequate anchorage for short fixed bridges with this type of attachment. When an anterior tooth is to be used as an abutment, the pinledge attachment or the three-quarter crown with accessory anchorage is usually employed.

The type of pontic is next to be considered. On the upper jaw, it is best to provide a pontic which will rest gently on the alveolar ridge whether in the anterior or the posterior region. For this, the ideal is highly glazed porcelain. This, of course, presupposes a sufficient amount of space between the ridge and the occlusal line to provide the necessary strength. If space will not permit this, it may be necessary to make the pontic of highly polished gold, with a Steele facing to provide the esthetic feature. If the bite is so short that this is not feasible, a gold pontic may be constructed, with a recess cut in the buccal side in which a porcelain inlay may be placed.

On the lower jaw in the posterior region, it is usually inadvisable to rest the pontic against the alveolar ridge. It is almost invariably superfluous from the esthetic standpoint, and the sanitation of the bridge will be accomplished with greater difficulty. In this region, the cleanable bridge is indicated. Here, a properly carved cast span is constructed which is so adapted to the abutment teeth that any food which may lodge under it is readily removed by the patient's tongue.

Fixed bridges are not always indicated, even when the span is short. Many posterior bridge cases are found in which the spaces are sufficiently short that fixed bridgework might properly be used, but in which the abutment teeth have not been attacked by caries. In these cases, it may be injurious to the pulps of the abutment teeth to do the amount of cutting required for the construction of inlays or three-quarter crowns. In these cases, it is preferable to make a removable bridge, using gold wire clasps for its retention, provided this can be accomplished without the buccal portion of the clasp being conspicious. It has been frequently urged that these clasps result in caries and that the case must eventually be made with an inlay or three-quarter crown. In the type of case to which I refer, the tendency to caries in the mouth is

so slight that the clasp is not contraindicated. In the event caries does occur, inlays may be placed as required, with the result that the vitality and health of the pulp are better conserved than would have been the case if a sound tooth had been cut for an approximoclusal inlay in the beginning.

The case presenting the usual indication for a removable bridge is one in which the space to be filled is so long as to require support by the alveolar ridge in addition to that afforded by the abutment teeth. It must be kept in mind that while the ridge under the saddle in removable bridgework takes a certain amount of the stress coming on the bridge, the abutment teeth also share in this stress. It is therefore unsafe to use abutment teeth even for removable bridgework if they have lost any considerable amount of their alveolar support. The prosthetist must plan his case so that there shall be an equitable distribution of stress between the ridge and the abutment teeth. In addition to the indications noted, removable bridgework may be used where posterior teeth are to be supplied and there are no posterior abutment teeth.

The method of retention of the removable bridge will vary according to the conditions found. The use of the gold wire clasp has already been mentioned for cases in which there has been no previous caries. This should always be used in conjunction with some form of occlusal rest. The tooth which already has had caries may properly have placed in it some form of internal anchorage. In deciding on the applicability of this type of attachment, the first requisite is that it shall not require cutting dangerously near the pulp. It is also important that it be placed within the natural circumference of the tooth in order to avoid injurious leverage. This injurious leverage may also be induced if the internal attachment is placed in a tooth which is tipped appreciably out of its normal alinement. The use of tipped teeth as abutments is always questionable, and, if they are used in removable bridgework, the clasp is recommended as the most suitable attachment in the majority of cases.

When the span of the removable bridge is quite long, as, for instance, one supplying four or five teeth, the lateral strain on the abutment teeth is considerable. In such a case, it is usually advisable to gain additional stability by carrying a bar on the lower jaw or a saddle extension on the upper jaw to a suitable tooth or teeth on the opposite side of the mouth. These teeth may have any suitable form of attachment, but it must be one which will limit excessive movement.

It has often been said that common sense must be the guide of the prosthetist. This is rather overworking a very good phrase. What the prosthetist needs is, first, an understanding of the fundamentals of periodontia, together with an appreciation of periapical pathology; secondly, he must have a natural comprehension of the laws of physics and their application to the mechanical requirements of the dental apparatus; and, thirdly, he must have sufficient esthetic sensibility to enable him to produce restorations which will please the eye.

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MANAGEMENT OF INFECTED TEETH IN THE PRACTICE OF GROUP MEDICINE*

By BOYD S. GARDNER, D.D.S., Rochester, Minn.

THE relationship between dentists and physicians of the Mayo Clinic is not unlike the relationship between other dentists and physicians, provided adequate examinations are made in their respective fields and due consideration is given to consultations.

All patients referred to the Section on Dental Surgery receive complete roentgenologic examinations regardless of the number of teeth present. However, it is realized that this examination is only one link in the chain of a dental diagnosis as teeth must be tested for vitality, and the history and clinical data must be considered.

The dental section functions as do other special sections in the clinic in that it first assists in the completion of general examinations. It then does the work of its specialty as indicated, but only with the approval of the clinician in whose section the patient is registered. In other words, patients are referred to the dental section for a report, and after the completion of the general examination, they may be transferred for dental treatment.

The general plan for the treatment of infected teeth in the Mayo Clinic has not been materially changed during the last ten years. More thorough examinations are being made as improved methods are instituted. Moreover, the surgical results have improved as the technic of extracting teeth has been refined, in an effort to improve postoperative conditions and to aid the dentist in carrying out his share of the care of the patient.

When the Section on Dental Surgery was first started in the Mayo Clinic, the physicians were not particularly interested in just what tooth or teeth were in question, but they did want a report concerning the degree of the infection. They also desired information regarding developmental defects, such as unerupted, impacted and malposed teeth. Thus, the dental sheet was made to coincide with reports from other sections in the clinic by indicating the degree of infection on a grading of 1 to 4, which is the same mathematical scheme as is used by the different laboratories in reporting their data.

The dental report sheet has been changed many times, to give more concrete information, and its size has been reduced so that it could be filed with the duplicate dental roentgenograms without crowding. It is now so arranged that a carbon copy of the part of the report that is of interest to the physician can be filled in without any possibility of error, and the remainder can be used for data that are of interest to the dental section only. All teeth, roots and questionable

*From the Section on Dental Surgery, The Mayo Clinic.

*Read before the Section on Oral Surgery at the Midwinter Clinic of the Chicago Dental Society, Jan. 15, 1929.

areas are still classified in two groups. In the first group are teeth which demand operation for local reasons, or in other words, those which should receive attention regardless of any possibility of relationship to systemic disease. This group, therefore, includes practically all impacted teeth with marked periapical infection, teeth with such severe gingival infection that local treatment is contraindicated, roots, residual areas of infection and foreign bodies. In the second group are listed teeth which should be removed in addition to those in the first group, if the systemic condition of the patient permits the removal of all dental foci. Accordingly, if indicated by systemic conditions, these two groups merge into one. It is the teeth in the second group that are occupying the attention of the dental profession particularly, because such teeth are generally pulpless and roentgenographically negative.

There has not been any material change in the number of roots found under full dentures, as the percentage continues to be about 33⅓. One in five (20 per cent) patients who have had extractions elsewhere have at least one or more roots left. It would appear, therefore, that there is still great need of improvement in the technic of extraction.

The method of numbering teeth in the clinic is different from that generally used, but it answers the purpose much better than any other. In dealing more directly with patients, it is much easier to make them understand if the upper left third molar is numbered one, and the lower left third molar is numbered seventeen, and so forth.

The films are mounted with the emulsion side protected, and patients can therefore be told to view the films the same as though they were using a mirror to examine their teeth. This idea aids materially in pointing out to them just which teeth are questionable. These mounted films are given to patients to be taken to their dentists, and accordingly are distributed in a very general way throughout this country and other countries. In this manner, dentists who never attend society meetings are reached and are aided in rendering better service to their patients.

Further knowledge of the pathologic aspect of pyorrhea shows that it plays the same rôle in focal infection as does infection at the apices of teeth. The condition of two patients who were observed in the clinic are illustrative of this point.

A man, aged 35, had suffered from arthritis for four years. The tonsils and all pulpless teeth were removed. The result of this treatment was very satisfactory, for a time, but the patient returned with another attack of arthritis, associated with acute iritis and prostatitis. After his first visit to the clinic, several pyorrhea pockets developed, and from them, a pure strain of streptococcus was cultured. Cultures from such pockets are not obtained as constantly as from apices of pulpless teeth. Several rabbits were injected intravenously and iritis resulted. The response to treatment of the prostate was not satisfactory, a result that is to be expected if dental infection is still present.

A woman, aged 45, had subacute infectious arthritis. Foci of infection other than pyorrhea were not present. One culture yielded a pure strain of streptococci. Cultures on animal inoculation were positive.

Many patients have pyorrhea which is apparently not sufficiently severe to warrant extraction of all teeth, and yet there is little, if any, hope of successful treatment. Many dentists care for this con-

dition just as they care for a mouth full of decayed deciduous teeth. They argue that, in the case of decayed deciduous teeth, it will not be long until the permanent set will erupt; and that patients with pyorrhea can stand the condition until they make their own decision regarding dentures. More and more, the need of early extraction becomes apparent in such conditions of pyorrhea, since the longer extraction is delayed, the less adequate will be the foundation for dentures. Moreover, the patients are subjected to infection which might be termed a load in the absence of any systemic condition traceable to a focus.

I am much interested in the surgical work in cases of pyorrhea such as is being done by Ward of San Francisco, and I have had excellent results in selected cases. Ward has popularized Black's surgical treatment of pyorrhea by the use of a surgical cement. After operation, he replaces, immediately, in excess, the soft and osseous tissue which has been cut away. The cement allays postoperative pain and offers resistance to tissue repair just as dentures do when inserted directly after teeth are extracted.

The insertion of dentures directly after extraction of teeth is growing in popularity and is looked on with favor by patients who are to lose their teeth. Tissues that have been traumatized by extraction react favorably when protected by dentures. The surgical preparation of the ridges makes it possible to construct more nearly uniform dentures, which should be considered as matrices for the regeneration of tissue. Therefore, the sooner they are inserted, the more nearly uniform will be their foundation. There is another factor in denture foundations. In certain cases, the process continues to disappear after dentures are inserted; while, in others, there is little if any absorption. With our present knowledge of bone changes, it is reasonable to believe that this rapid disappearance is caused by the same systemic condition as caused the loss before the teeth were removed; in other words, such a condition should be considered as a symptom of a general disorder.

Block anesthesia continues to be used in a large percentage of cases in the Mayo Clinic. It enables the operator to do better work, except in the easier type of extraction, and the risk is very much lower. It should be borne in mind that the loss of blood, loss of body heat or prolonged or uneven anesthesia, local or general, all share in the production of shock. The temperature of the average dental office is often so low that patients suffer ill effects especially when a general anesthetic is administered; and patients are often allowed to return to their homes sooner than they should because necessary conveniences are not provided in the dentist's office. The advantages, therefore, of hospital care can again be emphasized.

At the Mayo Clinic, we are fortunate in being able to do all our work at the hospital, with every facility offered to carry out operating room technic as in general surgery. This should not imply that all the patients are sick, as many are in as good physical condition as those who go to other dentists for treatment. It is, of course, impractical for the average dentist to extract teeth or do all such work in a hospital, but undoubtedly better results will obtain if more of the difficult operations are done under hospital conditions, as well as extractions of all types for persons who are ill.

Preoperative medication is not carried out as it has been found that any medicament that will destroy the organisms in the mouth will cause inflammation, a

condition that is not conducive to the best postoperative results. It is just as necessary to keep up the chain of asepsis in oral work as it is in other surgical procedures, and apparently the reason it is not carried out to a greater extent by the dental profession is the traditional one.

The value of the operating table over the dental chair for surgical work cannot be overestimated. Patients are better managed under either a general or a local anesthetic, and the position is advantageous to operator and to patient.

All deciduous teeth which cannot be filled without involvement of pulp should be extracted. This may appear radical, but it is justified by laboratory and clinical results. The objection to such a stand by some orthodontists is overruled, as it is believed that it serves the best interest of the child. It would be much better to engage the services of an orthodontist than to create in the mouth of the child a condition which might be the cause of a diseased kidney or heart.

There can be no fixed ruling regarding the extent of operative procedures feasible at one time. The general condition of the patient after the first operation, especially with regard to temperature, pain and elimination, as well as local results such as swelling, trismus and the condition of the wound, must all be recorded in order to stress the importance of not carrying the first operation too far.

The Section on Dental Surgery in the Mayo Clinic has started its eleventh year without a fatality, and I believe this record has been due largely to the fact that the work has been done under aseptic rather than antiseptic conditions, and to the careful management of acute conditions, the limiting of the number of teeth extracted at one operation and the more general use of local anesthesia.

The more difficult extractions of teeth are accomplished with greater ease as simpler mechanical methods are employed. The creating of space to facilitate extraction by the cutting of teeth, by the use of the chisel and mallet, is steadily gaining favor. This technic affords relief which, in principle, is the same as cutting away osseous tissue on the distal aspect of impacted lower third molars to facilitate their removal.

The open-view operation for the extraction of the majority of teeth continues to be in use. Even with present refinements, this operation is still receiving the usual amount of criticism from some of the so-called stand-pat members of the dental profession, but it is gratifying to learn that as the misunderstandings with regard to the exact nature of the operation are cleared up, it is becoming more popular.

The postoperative roentgen-ray examination as a routine procedure is the impetus which, if used, will increase the popularity of the operation, as it will stand the test of such examination. This reference to technic does not imply that this method of extraction should be used in every case, as each case is a study in itself, and many teeth are still being simply "pulled," and satisfactory results obtained.

Indications for curettement of sockets following the extraction of teeth with infection at their apices is not determined by roentgenologic evidence alone. The sense of touch should guide the operator, and normal tissue should not be disturbed. When the granuloma is still attached to the extracted tooth, at least slight curettement is indicated to insure elimination of all diseased tissue. Extra care must be taken if the tooth to be removed has been in disuse for some time because the associated osseous tissue is so

soft that the operator cannot judge from touch alone.

Curettement may consist of very delicate or quite severe manipulation with an instrument, this depending on the particular case. Curettement is not indicated in acute conditions. After a month or two, if the patient is reëxamined and the roentgenogram or clinical evidence indicates retained diseased tissue, curettement is indicated, and can be done with much less risk.

We continue to find such a large percentage of roentgenologically negative pulpless teeth to be infected that all must be considered potential foci of infection and should be extracted if the condition of the patient requires the elimination of all dental infection. In other words, the safety of leaving such teeth depends on the defense or resistance of the patient, rather than on the ability of any dentist to keep them from doing harm. It is apparent, therefore, that one cannot be satisfied with an occasional roentgen-ray examination of pulpless teeth which are allowed to remain. Such patients should have a complete general examination in order to permit the determination of the status of these questionable teeth.

Work in the bacteriologic laboratory continues to stress the importance of questioning all pulpless teeth. In a recent study, it was found that 94 per cent of teeth shown by the roentgen ray to be diseased gave positive cultures of streptococci, and 84 per cent with negative roentgenograms gave cultures of streptococci. The recent investigation of pyorrhea pockets emphasizes the need of more work in this respect. The method of obtaining a dental specimen for laboratory study is so controlled in each patient that the results of such investigations cannot be questioned from that standpoint.

PREOPERATIVE PREPARATION, TREATMENT AND MEDICATION*

By STERLING V. MEAD, D.D.S., Washington, D. C.

THE time to arrange for a successful operation and a good postoperative result is in the preoperative stage. No matter how successful the operation, a postoperative complication of any nature is very discouraging.

It is becoming more apparent to all of us that there is a great advantage both to the patient and to the operator in close cooperation with the physician. A thorough physical examinatiton by the physician may save a great deal of embarrassment in what appears to be even a simple extraction. It is very unwise to operate in the presence of such conditions as diabetes, specific diseases, Vincent's infection, acute infections, septicemia, anemia, leukemia, hemophilia and circulatory and other systemic disorders, without proper control or preparation.

It would be a great advantage, if it were practicable in every case, to determine the hemoglobin content, red cell

*Read before the American Society of Oral Surgeons and Exodontists, Minneapolis, Aug. 17, 1928.

count, white cell count, differential count, clotting and bleeding time, and blood pressure, and to have a urinalysis and a serologic test. One of many similar cases that I have observed will illustrate the advantage of this.

REPORT OF CASE

Mrs. C, aged 75, had previously come to me for the extraction of septic teeth, three to four being removed at each sitting. She had recovered promptly without any apparent inconvenience. About one month later, she returned for the removal of two incisor teeth. They were not sore and were removed easily. About two days later, I was called in the evening by the husband, who was in a very bad frame of mind because, as he said, his wife had been to my office for treatment and had steadily grown worse. I called to see her and found her mouth in fine condition, but she appeared to be in a dazed condition and apparently toxic. A physician who had been in to see her that day found no trouble and stated that the case was one for the dentist. I advised giving her plenty of liquids and orange juice, and suggested calling the physician again in the morning. The next morning, the patient called another physician, and he immediately called me, stating that the patient was in a diabetic coma. If this patient had died without the discovery of the diabetic condition, the extraction would have been blamed.

I feel that even the extraction of teeth is not a simple matter, but one deserving the greatest skill, training and experience. There are many people losing their lives because of neglected mouths, and, too, many people start to care for the mouth when it is too late. It naturally follows that the removal of teeth is often followed by, or associated with, other systemic disturbances, occasionally with fatal termination. Death may follow any extraction from other causes than unskilful attention, but in order to keep unfavorable postoperative results at a minimum, the aid and service of the physician should be sought. Cooperation between the physician and dentist is imperative. Intelligent and successful mouth surgery cannot be practiced without some knowledge of the patient's physical condition.

The mouth should be clean and in as good sanitary condition as possible. Loose dental work, plates, etc., should be removed. The mouth should be sprayed with a pleasant cleansing solution. When a local anesthetic is used, it is well to spray the tissues with some pleasant solution containing anesthesin or butyn, eugenol, etc., to aid in obtunding the terminal nerves and preventing pain during injection of the drug.

The anesthetic should be chosen after considering the requirements and the type of the patient. The patient should be placed in a comfortable position in the chair. I have found that children and very nervous patients react more favorably when they are reclining on a table. In many cases under local anesthesia, it is well to cover the eyes of the patient with a towel.

Whether we are working with a local or a general anesthesia, gauze packs should be placed in the mouth to keep mucus and saliva out of the wound as far as possible, to help take care of the blood and also to act as an aid in preventing particles of any kind reaching the throat. The gauze also aids in preventing mouth breathing in general anesthesia. When procain is used, the amount of epinephrin should be regulated according to the type of patient. The use of spirits of ammonia, from one-half to one teaspoonful, immediately after the injection of procain, will, in many instances, prevent syncope and other unpleasant symptoms. When in the dental chair, the patient should be tipped back to a reclining position after injection of the anesthetic.

Whether local or general anesthesia is to be used, the patient should be pre-

pared for the operation. In the more complicated surgical cases, the following plan is advisable: Two days prior to operation, from one to two teaspoonfuls of milk of magnesia is given, and the patient is put on a green vegetable diet with orange juice. In the morning of the day of the operation, a soda enema is given, and nothing is taken into the stomach, except a glass of orange juice two to three hours before operation. For the average case of removal of teeth, the only preparation necessary is to see that the stomach is empty, except for a glass of orange juice taken three or four hours before operation. I feel that, in this way, the patient is not subjected to the hunger pains of an entirely empty stomach, and also that the preoperative preparation and the taking of the orange juice are an insurance against acidosis. In this way, nausea is practically eliminated. After operation, the food for that particular day is limited to orange juice, lemon juice or grape juice.

Preoperative medication is but occasionally necessary. In the extremely nervous patient, or in the case in which an extremely difficult case would subject the patient to shock, I prefer one-sixth grain morphin sulphate, repeated in thirty minutes, when necessary. This works well with either local or general anesthesia, but is usually not suitable for ordinary dental routine. When this method is not practical, phenobarbital (luminal) one-tenth grain, for children, 1½ to 3 grains for adults; triple bromid; barbital, bromural, etc., may be administered.

When it is necessary to correct the bleeding or clotting time of the blood as a preoperative measure, this may usually be accomplished by 1 teaspoonful (40 grains) calcium lactate, three times a day for from four days to one week; the use of the actinic ray light beginning three minutes at 18 inches and increasing one minute each day; 1 teaspoonful of cod-liver oil and gelatin with meals. In some cases, I have seen improvement from intramuscular injection of coagulen. In the aggravating cases and those not responding to other treatment, the blood should be typed and an intramuscular or intravenous injection of blood should be given.

There can be no standardized method in the care of painful or sore teeth or teeth with acute symptoms of infection. Each case is necessarily handled according to the existing conditions, and judgment and experience on the part of the operator are necessary. In general, it may be stated that it is usually best to attempt to tide the condition over the acute stage or reduce the acute symptoms before operation. A sore throat, a cold, a swollen lymph gland, a sore tooth and an inflamed gum are all danger signals, and care should be exercised in operating in their presence. In many cases, an attempt at treatment to reduce the acute condition will fail, but at least Nature may be given an opportunity to form antibodies and localize the infection.

I rarely attempt to remove impacted third molars when they are sore or acutely infected, because of the danger of involvement of the throat, peritonsillar abscess, etc. The tonsils should always be examined carefully, as many cases of postoperative complications are due to the stirring up of infection in the presence of infected tonsils. The cooperation of a rhinologist often is of the greatest value in throat conditions, as well as in complicating conditions of the maxillary sinus. There is danger in removing teeth bordering the maxillary sinus when they are acutely infected, because of the danger of sinus infection, pansinusitis

and brain abscess, as acute infection may travel fast.

The third molar area is best treated by cleaning under the flap of soft tissues with hydrogen peroxid, irrigating with mild antiseptic solution and placing a pledget of cotton under the flap, saturated with hexylresorcinol S. T. 37 or 5 per cent mercurochrome, and seeing the patient twice each day, if necessary. It is well to defer the operation until all the soreness disappears. In many cases in acutely infected lower third molar regions, it will be found that an upper third molar is elongated or out of alinement and irritating the gum over the lower molar, when the mouth is closed. In these cases, it is well to remove the upper third molar first and defer removing the lower molar until later.

Any soreness, swelling or uncomfortable condition about the lower third molar, with trismus, should suggest the possibility of a peritonsillar abscess, and the throat and soft tissues should be swabbed carefully and early incision be made when indicated.

Soreness in the mouth may be relieved by the use of a hot salt and soda mouth wash. Cold packs applied to the face may prevent swelling. When a swelling localizes, hot magnesium sulphate packs are indicated. Localized swellings or infections in the mouth should be incised promptly for drainage. Enlarged tender lymph glands, in the presence of soreness or an acute infection in the mouth, may be treated likewise. The danger of removing teeth in the presence of acute conditions does not arise from the possibility of bone disturbances, but more from possible cellulitis, septicemia and soft tissue involvement. I would not hesitate to remove teeth during bone disease whenever indicated, but all traumatism should be avoided.

In many cases of soreness, swelling or acute conditions, there is no good reason why the tooth should not be removed when it feels soggy and pus is around it, as this will assure the best drainage. The removal of teeth is more to be feared when drainage of the acute area will not result but the infection will simply be stirred up and a larger field for the infection be produced.

DISCUSSION

Frederick F. Molt, Chicago, Ill.: If we all had the opportunity Dr. Mead has spoken of, of seeing our patients and providing preoperative care, we would be at an advantage. However, we are at a disadvantage in our practice because situations present themselves in which we must make quick decisions and must necessarily operate under circumstances that are not entirely ideal. The precautions Dr. Mead has spoken of are well taken, and I heartily agree with him in everything he has said.

A. B. Vastine, Danville, Pa.: There is a very important point in this paper that has been touched on but has not been emphasized to the extent that its importance warrants; that is, preoperative medication of the gingival trench, based upon smear findings. In acute conditions, the bacteriology of the gingival trench is extremely important. This was hinted at when Dr. Mead spoke of placing a swab of medicated cotton under the flap of the third molar to prevent abscess formation. If a smear is taken and subgingival medicatiton instituted with a specific or near-specific for each organism entering into the infection, we will not only be able to control postoperative infection but also to prevent other reactions such as, for instance, a peritonsillar abscess, or any infection entering into this question of preoperative preparation. I know of practically no case in which removal of teeth is justified unless the patient has been given the benefit of thorough preoperative medication of the gingival trench, not only for the tooth involved but also for the whole mouth.

Thomas Conner, Atlanta, Ga.: A case which I had recently will further stress the importance of the valuable point that Dr. Mead has just brought out. A young man came in with the usual symptoms of an

acute infection of a mandibular third molar. He came in on Monday preceding the Fourth of July, on Wednesday. I gave the usual preoperative treatment, very much in line with Dr. Mead's treatment. On Tuesday, the patient came in, insisting that I remove the tooth because, he said, he was very toxic and had pains in his left shoulder and right knee, which he was quite positive came as a result of the infection about this tooth. The treatment of the preceding day was repeated. I told him that I would like to have him see his physician. This he did Tuesday noon, and was sent to the hospital. I did not know that he had gone to the hospital, but on Wednesday morning, I was informed that he was there and would expect me to come by to see him some time during the day. The physician saw him on Wednesday about 11 a. m. I saw him about 3 p. m. and he was still insisting that I take out his third molar. On examinatiton, I found that the acute symptoms had subsided, but he had a temperature of 101 and was perspiring profusely. His nurse told me that she had changed his pajamas three times during the day. I asked him if he had had any wounds from which we would expect tetanus infection, but he had had none. I had never seen a case of tetanus and was not positive of the early symptoms so went to the hospital library to learn what they were. The only symptoms that he had were the profuse perspiration and a rise in temperature. I called his physician and told him that I thought our patient had tetanus. At 7 p. m., he had all the symptoms of a well defined case of tetanus and the usual treatment was started immediately. At present, the patient is quite normal. If I had extracted that tooth on Monday, most of my acquaintances would have given me credit for having caused lockjaw. I think it is most important that we have these cases, especially of the third molars, in a chronic or semichronic condition before we operate.

Dr. Mead (closing): Dr. Vastine brought out the importance of the treatment not only of acute conditions of the gums, but of chronic conditions as well. I heartily agree with this. In every case, whether we have acute symptoms, or apparently a tooth that can easily be removed, our first thought should be prophylaxis of the mouth. We should use every care to put the mouth in as nearly perfect condition as we can before we attempt even simple surgical incisions. There are many cases, such as the case Dr. Conner has presented, which show us the advisability of handling acute conditions cautiously. The patient is in great pain, there is discomfort and great pressure is brought to bear on us to give relief, but a great many of us have been through experiences that have led to regret, and we learn to handle acute conditions very cautiously. Along the line of tetanus, I saw a tetanus case that developed from a little scratch on the hand, arising from cutting and handling meat. It is possible in some cases to develop tetanus for apparently no real reason. Dr. Morris has asked me to speak about the incisions of acute abscesses. It is quite a prevalent practice among many men to treat a lower third molar before referring the case to a specialist. The condition becomes acute, and they lance the abscess. In many cases, this is done inopportunely, and when the case is presented to the specialist for treatment, there is more or less cellulitis and spreading infection. Lancing over lower third molars is an unsafe procedure, except when it is indicated. If we have localization, or beginning localization, by a small incision, and slight opening made with a hemostat, and not too much traumatism, we may get drainage and clear up the case, and it is absolutely indicated. But because of soreness, or the presence of Vincent's, or some other acute infection, lancing may simply lead to the development of a very virulent stage.

COMPREHENSIVE REQUIREMENTS IN OPERATIVE DENTISTRY*

By JAMES M. WALLS, D.D.S., St. Paul, Minn.

A STANDARD of high attainment in dentistry was never more earnestly sought than at the present time. Observation of results attained by efforts, over the past twenty-five years, to save the teeth of our patients have been far from satisfactory. A study of the mouths of people in middle life reveals such a large percentage of missing teeth that a rather gloomy picture is presented. It was with the hope of calling attention to some features in the realm of operative dentistry which seem insufficiently emphasized and which have a bearing on this unfortunate condition that I chose to discuss comprehensive plans in procedure.

New methods in science have produced definite knowledge of conditions that, but a few years ago, were masked in obscurity. Civilization of the higher order owes its existence to the free development of the sciences. Its destiny will rest upon the application made of science to our social fabric and its adaptation to our physical welfare. The field of medicine has long been held a unit in the group of advanced sciences, and some ascribe to dentistry the place of a specialty in medicine. While it is true that the basic principles of dentistry rest on health service, there is so much of the extra physiologic technic woven into its practices that the more essential considerations are often overlooked by the public. Its contributions to the prevention and elimination of disease place it squarely in the field of medicine, or denominate it a science complimentary to medicine. Operative dentistry is charged with the responsibility of maintaining the teeth in normal function, through specialized effort.

To a group whose efforts and training are mustered in the cause of tooth conservation, their appalling loss presents itself as an open challenge for improvement. Through human enterprise, great progress has been made in scientific achievements. These have resulted in marked changes in our social structures and habits of living. Physiologic controls of the body have been so upset by these changes from primitive customs that we find the highly civilized groups are afflicted with diseases not common among the uncivilized. There is none more prevalent than caries of the teeth. Persistent efforts through physiologic chemistry have been made to ascertain the exact causes of this disease. Results have been obtained through changes in diet and other habits, but not to a degree that permits us to relax our program of treatment by mechanical means.

Through the advent of broader conceptions, specialization in dentistry has been developing and is beyond question increasing our total fund of knowledge. Specialization tends to produce gaps that

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are not always covered. In this situation, we find operative dentistry failing at times to assume responsibilities broad enough to obtain the best ultimate results. It is the distinct function of this branch in practice to advise patients to maintain normal function of the teeth and, when defects occur, to instigate such procedure as seems warranted to produce efficiency. Almost at the outset of life, we are met with the requirement of attention to the teeth. Experience reveals more forcibly each year that the degree of health found in the mouths of adults indicates the comparative degree of proper attention given the teeth in childhood.

There are three major considerations in the treatment of defects of deciduous teeth and arches: 1. The conservation of the teeth for the function of mastication. 2. The maintenance of the deciduous teeth as a protection to the permanent tooth follicle, a stimulation to bone development and the preservation of spaces for the proper eruption of the permanent teeth. 3. The prevention of systemic disturbances and lowered resistance consequent to focal infection.

The first two factors are of such evident importance that indifference to their requirements places neglect squarely in the light of malpractice. Frequent visits to the dentist, the help of parents, careful protection of the pulps, the restoration of teeth by as nearly permanent means as the case in hand will permit and advice on diet for protective immunity are the best safeguards. Dr. Rhobotham of Chicago has given an excellent operative outline for the care of deciduous teeth, and Percy R. Howe's treatises offer valuable suggestions on diet. Ability of the child to masticate with comfort has a marked effect in the development of the jaws and exerts an important influence on metabolism. The early loss of deciduous teeth through disease suggests the use of appliances to retain the spaces for the proper eruption of the permanent teeth. Simple appliances for this purpose may be made and should always be so constructed as to provide occlusal contact for the opposing tooth, to prevent its extrusion into the space in the opposite jaw.

Recent advices on this treatment of cases have been presented by Dewey, Steadman, Sippy, Earnst and other orthodontists. The responsibility should rest on the general practitioner, and if the case is not referred to an orthodontist for treatment, it must be taken care of by the dentist in charge.

Many cases arise in patients under 12 years of age, in which the first molars are badly broken down or have exposed pulps. It is often advisable to extract such teeth prior to the eruption of the second molar, the operator allowing it to advance into a position of fairly good occlusion. Retaining appliances and bridges are thus avoided, the hazard of possible failure of maintaining teeth indefinitely being removed. The extreme importance of such cases makes it imperative to consider well all circumstances, including orthodontic factors.

It is highly important, therefore, that operative dentistry inform itself in the simple practices of orthodontia. The ability to foresee changes that are likely to occur where irregularities exist often alters our immediate procedure and affords advice of incalculable value to patients. Many of the more difficult problems that we are called upon to handle in the adolescent period of life result directly from neglect in early childhood.

The third consideration, that of infective susceptibility from foci about de-

ciduous teeth, is a subject that I am convinced has not been fully enough considered by the dental profession. There is at present a decidedly upward trend in this direction, but a prevalent opinion that child resistance is adequate to offset any serious consequences is often revealed. Intimate contact with several pediatricians of recognized ability and generous conceptions impresses me with the fact that they are extremely apprehensive regarding focal infections from the mouth. One authority in this branch of medicine states that it is difficult to take those measurements of young children that can be obtained from adults respecting resistance and susceptibility to disease. His experience leads him to a firm conviction that these foci are a factor in causing certain physical disabilities in children such as myalgia and secondary anemia.

Thus, the period of childhood presents many obstacles to operative dentistry and often calls for rare judgment in procedure if future conditions are to reveal no embarrassing results. During the adolescent period, diseased and functionless teeth, those presenting unfavorable relations with others and the development of spaces due to noneruption, present many interesting problems in the care of mouth conditions.

When the proximal surfaces of any teeth in young people are affected by caries, permanent restoration should be the paramount consideration, provided the development of the teeth and the health of the patient permit. This demand calls for a filling material that will be resistant to chemical and physical changes. Disastrous results due to the wasting away of fillings is noticed in the shifting of teeth, disease of the peridental tissue, closing up of the arches and recurrent caries.

In the case of cavity preparations in either the front or back teeth, sufficiently extended margins may be laid without excessive waste of dentin; nor is it necessary in the case of front teeth to reveal gold conspicuously, if that material is selected.

When cement is used as a temporary filling, spaces should be maintained by inserting with it some material such as a fragment of porcelain. It is of the utmost importance in making all proximal fillings that convex surfaces be so constructed that the food carried over them will be directed away from the gingival embrasures. In making fillings for proximal surfaces in bicuspids and molars, properly shaped marginal ridges and occlusal grooves for the escape of food over the lingual and buccal surfaces insures easy maintenance of healthy conditions, provided the contacts are firm and surfaces smooth.

The greatest bulwark against caries is to assist nature in keeping the mouth as nearly as possible automatically hygienic. Dietary suggestions are often a great help, but in this matter we encounter fadisms which have insufficient biologic and physiologic background to support them. In making restorations in or on the teeth, the following principles are of primary importance: conservation of the tooth for esthetic reasons and against fracture; protection of the pulp from undue irritation, and the establishment of form to produce efficiency in service and maintain the health of the surrounding tissues.

There has been a trend of late years, and particularly since the introduction of the gold inlay, to overextend cavities in both surface dimensions and in depth. A reaction to this has set in, and I am inclined to feel that there is some danger of the pendulum's swinging beyond

reasonable adjustment, to the other end of its sector.

The Class II type of cavities, more than any others, introduce the likelihood of fractured cusps or degenerating pulps. More especially is this true in that type of teeth presenting long cusps. As these wear away on the incline surfaces, the wedging stress during occlusion increases. Deep gingival walls are not necessary in preparation for stable restorations of either the gold foil or inlay type of fillings. In the desire for strength, there has been commonly followed an overextension of depth in the gingival area; whereas, with proper resistance form and well proportioned occlusal anchorages, shallow gingival walls are more desirable. Such precaution conserves greatly the strength of the tooth and reduces irritation to the pulp.

The occlusal preparation of all cavities of this type of teeth with long overbites should be made as shallow as possible and the surface of the filling material raised slightly above the original tooth surface to produce the necessary volume of gold. An opposing cusp may be slightly ground to relieve occlusal stress. It is always good practice to protect the cusps of weakened teeth by covering them with a sufficient volume of filling material to withstand stress; a rule that applies in most cases to mesioclusodistal, and often to large mesioclusal distoclusal cavities in bicuspids; the lingual cusps being the ones most vulnerable to fracture. The study of occlusion is fundamental and should in all cases be carefully observed, whether for a single tooth or for the entire arch. If there is much reparative work to be done in a case, and especially those revealing broken contacts, casts will be found an expedient means of study. A detailed routine of careful observation requires little time and assures results that are otherwise unattainable. Broken contacts and narrow spaces between teeth are a common cause of caries, as well as of irritation and infection of the peridental tissue, owing to food impaction. When spaces are wide, shifting of the teeth should be prevented by fixed bridgework, where possible. Where back teeth move gradually under stress, there is always a concomitant closing of the bite. There are many cases, usually in middle life, in which opening the bite is the only assurance against constant attention to the teeth or gums.

The importance of pulp protection was never more fully realized and thus we are warned against undue removal of dentin. Longitudinal sections of the teeth, especially of the young, reveal little thickness of dentin between the surface and pulp at the cervical line of the tooth. This suggests extreme caution when decay extends deeply gingivally. The protective agency of cement in all cases of deep cavities and in many others that seem not so deep helps to avert much trouble. Cement cannot be used with impunity. Where cavities of extreme depth occur or the dentin is particularly sensitive, a protecting film of varnish or other material is desirable. The cementation of inlays or crowns should be preceded by the utmost care that all foreign matter has been previously removed. Complete dessication of a cavity is discouraged by some chemists. The use of phenol as a coagulant is advised and seems clinically to have merit.

It is felt by some of my confrères that a cause of discoloration and disintegration of cement noticed occasionally under inlays is the contamination from mucin or other organic matter. The subject of the cements is one that should receive much more consideration than it

does, owing to our great dependence on it.

There are many subjects tangent to operative dentistry a partial knowledge of which seems necessary for a complete working basis, but one is not capable of mastering them all. It has been my endeavor to avoid discussing technical procedures, but to present a principle and to outline some of the features that have a direct bearing on permanent maintenance of the teeth. We recognize the demand of operative dentistry in exactitude and skill, in the display of a knowledge of physics and an expression of art, and we are beginning to realize the great importance of a broader biologic foundation that we may better calculate the relationship between the conditions that we observe in the mouth and the organic functioning of other parts of the body.

A situation resulting from the ferment in medicine over the question of systemic disturbances due to devitalized teeth is losing its acuteness through a better understanding between medicine and dentistry. This was a situation in which science and moderation parried with ignorance and radicalism. The leaven has been working until much of the violence has disappeared, and, in its place, cooperation between a dentist better trained biologically and a more rational and heeding medical profession is under way.

There is no more urgent need in dentistry than a greatly increased percentage of high grade restorative work. A degree of skill to meet reasonable requirements could be arrived at by the great majority of the profession if there was the stimulus to put effort into the task. It is distressing to see so much work of an inferior character done, and to realize often that it would have been much better for the patient had the work never been attempted.

Such results reduce tremendously the standards of American dentistry, and explain in great measure why such a large percentage of people who reach the age of from 40 to 50 have few natural teeth.

In the atmosphere of high standards in dentistry, where the best in our profession are working earnestly and skilfully to produce an enduring result and are seeing patients frequently enough to prevent any extensive development of disease, we find a picture of more pleasing character. These standards can be established only where the operator trains himself so thoroughly that any type of procedure (including peridental, pulp and filling operations) can be executed skilfully.

It is to be hoped that, through research, easier and cheaper methods of treatment will be discovered, whereby the great mass of people can be benefited in prevention of the loss of natural teeth.

THE IMPORTANCE OF TISSUE CHANGES UNDER ARTIFICIAL DENTURES*

By WALTER H. WRIGHT, D.D.S., F.A.C.D., Pittsburgh, Pa.

SINCE dentures were first made, our profession has been confronted with the complicated changes of the denture supporting tissues. The general term "tissue change" will be used, in this particular, to describe resorption or "shrinkage" of the alveolar residual ridges which occurs after the natural teeth have been lost. What dentist is not familiar, to his sorrow, with the results of tissue change? Why do the most nearly perfect restorations occasionally become loose and unserviceable, after being worn by the patient for but a short time? Are we certain that the denture may not be contributory to resorption? Why do denture supporting tissues change? How does this change affect the dentures, the tissues and the patient? How can tissue change be prevented? These are the questions which I shall discuss, although not exhaustively; for I shall confine my paper to the practical side of tissue change.

A method has been devised whereby the amount of tissue resorption can be measured. This method involves the selection of a patient who has previously lost several posterior teeth on each side, together with a central or lateral incisor from the anterior region of the maxillary arch. The patient should not have worn a partial denture. The infra-orbital margin of the maxilla is accepted as a reference base for the subsequent orbito-oral measurements. A special slide tube caliper is used for these measurements. The distance from the infra-orbital margin to the crest of the ridge in each edentulous space is measured and these measurements are recorded on a suitable form and filed for future reference. After the remaining teeth have been extracted, similar measurements are made in the same locations of the ridge.

Additional measurements are taken at monthly intervals after the dentures have been inserted. These measurements, when compared with the original ones, are progressively shorter, indicating the amount of resorption which occurred during the intervening time. The total amount of resorption of both maxillary and mandibular ridges is determined by subsequent measurements from the infra-orbital margin to the base of the chin, while the dentures are in the mouth. Resorption of from 1 to 3 mm. in the maxillary ridge, within a period of two months after insertion of dentures, has been recorded.[1] A prolonged investigation will undoubtedly show a gradual slowing up of this process until the maximum resorption has occurred.

Oral measurements of the height of maxillary and mandibular ridges show many that are between 15 and 20 mm. high; likewise, many that are less than

*Read before the Section on Full Dentures at the Midwinter Clinic of the Chicago Dental Society, Jan. 15, 1929.

1. Unpublished denture record charts, Department of Clinical Prosthesis, School of Dentistry, University of Pittsburgh.

5 mm. high. This variation suggests that a total of 10 mm. of resorption may occur in the average ridge; although this may be more or less for the individual patient. Time does not appear to be a constant factor in resorption. One would naturally suppose that patients wearing dentures for a short time would manifest little resorption, while those with long denture experience would show a great amount of resorption. Strictly speaking, this is not true, for some patients who have worn dentures for many years show much less resorption than others who have had but a few years of denture service. This variation can be accounted for only when the tissues, the dentures and the physical condition of the patient are taken into consideration. The reaction of tissues and the patient are physiologic in nature, and present a complex problem which is beyond the scope of this paper. The dentures play an important part in the process of tissue change, which will be considered as we proceed.

The difference in structure and function between natural teeth and dentures is well known, and I shall discuss only one phase of this dissimilarity. The alveolar process with a complement of natural teeth is accustomed to an internal pressure; that is, one applied from the center of the alveolar structures which tends to expand the alveoli. The alveolar residual ridge with dentures is quite different, for it is subjected to pressure from the outside which tends to compress both the osseous and the soft tissues. Force applied to natural teeth is distributed to the dense connective tissue fibers of the peridental membrane, which are designed to withstand this pressure. Pressure applied to dentures is distributed to epithelial tissue, which undoubtedly reacts proportionately to the amount of pressure and the manner in which it is applied.

The effect of even a slight amount of pressure upon epithelial tissue may be observed under the ring on one's finger or under the bridge of one's spectacles. The tissue gradually atrophies until a point of physiologic tolerance is reached. Microscopic sections of flabby tissue found under an upper denture show hypertrophy of the investing tissue, with a structure resembling hard fibroma.[2] A moderate amount of round-cell infiltration is present beneath the epithelium in areas where the epithelium came into contact with the denture. This is probably only an inflammatory reaction to pressure from the denture. There is apparently no evidence of infection. Atrophy of the denture supporting tissue is due, no doubt, to pressure.

In order to emphasize the important part played by dentures in resorption, I must differentiate the two general types of tissue change which are met in daily practice. Natural atrophy is common to all patients who have recently lost their natural teeth. The alveolar process resorbs rapidly at first, and then resorption gradually decreases until the tissue reaches a state where further change is slow and regular. This atrophy is influenced by blood, circulatory, connective tissue and, possibly to some extent, by mechanical factors.[3]

In the second type, I prefer to consider the mechanical factor in resorption separately, since this factor is usually present with dentures. When partial or complete dentures are worn, they possess, from the mechanical standpoint, either

2. Moorehead, F. B., Dewey, Kaethe: Pathology of the Mouth, Philadelphia: W. B. Saunders Company, 1925.

3. Leriche and others: Physiology of Bone, St. Louis: C. V. Mosby Company, 1928.

balanced or unbalanced occlusal relations. I wish it were possible to establish a definite standard by which the effect of unbalanced occlusion could be accurately predicted. This seems impossible on account of the difference in physical resistance found among patients. Have we not seen mutilated ridges associated with fair occlusion, and excellent ridges even when unbalanced occlusion was present? These exceptions, however, are not the whole story. Let us hear the evidence of those who wear dentures.

Here are 200 patients with new dentures; they have never worn dentures before. Sixty-eight per cent of them have unyielding tissues covering the ridges; while 32 per cent have yielding tissue conditions. There is none with mutilated flabby tissues. Let us examine 125 patients who are experienced denture wearers. Twenty-one per cent of them have unyielding tissues and 79 per cent have yielding or flabby tissue conditions. Something has happened since these patients received their first dentures. Conditions are almost reversed in these two groups.

What effect have single dentures on the ridges? Three hundred and fifty patients who have worn full upper dentures while they gradually lost their own lower teeth will answer this question. Twenty-two per cent of them have unyielding or bony tissue conditions on the upper ridge, and 78 per cent have yielding or flabby conditions.[4] Those who retained a few lower anterior teeth invariably developed a flabby upper anterior ridge, which made it difficult and often impossible for the operator to construct a new denture with stability and balanced occlusion. Practically every set of old dentures that was examined had the wrong jaw relation and was in unbalanced occlusion. Every single upper denture functioning against a few remaining lower teeth was in unbalanced occlusion. If it is admitted that tissues will resorb even under dentures which have the correct jaw relation and balanced occlusion, what then can be expected from dentures which, as a result of resorption, have sunken out of occlusion? These data seem to be an indictment against dentures, and yet the dentist cannot correct the conditions unless his patients return to avail themselves of his services. Is the patient not responsible for his own negligence, especially after he has been warned concerning the effects of resorption?

The results of tissue change may become increasingly far-reaching and destructive, until the entire masticatory mechanism and even the patient's health is involved. This dangerous cycle of reactions affects, in turn, the occlusion, the health of the tissues, retention, esthetics, anatomic structures and the physical and mental condition of the patient.

When atrophy occurs under newly constructed dentures, the occlusion is first disturbed; and, invariably, unbalanced occlusion results. The posterior teeth soon sink out of occlusal contact, owing, apparently, to the burden of mastication placed on the molars and bicuspids. The anterior teeth come into premature contact and the retrograde process begins. Pressure applied to localized areas of the residual ridge through unbalanced occlusion causes atrophy. As resorption continues, various areas are in turn affected by this unequalized pressure until unbalanced occlusion becomes a powerful agent in the destruction of denture supporting tissues. As resorption progresses, the lower jaw closes and swings forward, an element of protru-

4. These figures were compiled from the denture record charts for the years 1925, 1926 and 1927.

sion resulting which complicates occlusal relations. This process may continue until a "pipe stem" mandible fractures under the stress of mastication, or until the antrums are involved by atrophy of the maxillae.

While these serious changes are taking place, the investing membrane of the ridges may be involved. Usually, although not always, the soft tissues become inflamed. This may be the cause of the so-called "burning sensation" under a denture, or the more common malady "rubber sore mouth." Recent experiences with these conditions impress me with the great part played by unbalanced occlusion in disturbances of the soft tissues. The correction of occlusal relations may remove, as by magic, all the trouble with which a patient has been inflicted for years. Quite frequently, the tissue becomes flabby; which seriously interferes with the stability of the denture and may call for surgical removal.

Why does not this tissue under such unfavorable conditions develop malignant neoplasms? When the form of supporting tissues changes, the retention of dentures is affected. No denture, in reality, maintains its original retention. Sooner or later, the tissues change and the denture becomes loose. This change frequently occurs so slowly that the patient becomes accustomed to the progressive loss of retention. I have often marvelled at the ability of patients to wear dentures which were held in position by the tongue.

Change of dentures in relation to the lips and lower third of the face has a profound effect on the patient's appearance. The chin approaches the nose, the lips atrophy and become wrinkled, the cheek becomes sunken, the nose develops a characteristic downward curve, and often the patient cannot be recognized by his old friends. This is a great price to pay for neglect.

These changes in anatomic structures are serious since they cannot be corrected. New dentures may be constructed, balanced occlusion may be restored, but the effects of neglect will invariably reduce the efficiency of subsequent restorations. I have never observed the process of an edentulous ridge being rebuilt, but I am inclined to believe that even though the process of resorption may be retarded, new ridges cannot be developed.

How does the patient tolerate the process of tissue change? I have seen some who have worn loose fitting, very poorly occluded dentures without apparent discomfort. It is a pleasure to make new dentures for such a patient and to have them explain afterwards that they now know what it means to have a well fitting denture with teeth that actually cut food.

Not all patients are so fortunate. Many are very sensitive to these changes. I recall one patient who developed such pain in the region of the temporomandibular joint, in response to unbalanced occlusion, that she questioned the advisability of living under these conditions. The correction of occlusal relations gave relief in less than one month. Another patient, who had suffered with soreness of the mucous membrane under dentures for over two years, was relieved in two weeks by the correction of the occlusal relations. Each patient is different and it is difficult to predict the reaction to the sequelae of unbalanced occlusion.

Is it not safe to assume that unbalanced occlusion is one of the most important causes of tissue change? In the light of this discussion, I cannot close without stressing the necessity for balanced occlusion in denture restorations.

Balanced occlusion must be maintained throughout the life of dentures. We must impress on our patients the importance of frequent visits for examination, adjustments and correction of restorations. Patients must not be dismissed with the belief that their dentures will last the rest of their lives. Five years may be the limit of a denture's usefulness. There are continuous advances in the form of teeth and arrangement of them in favorable occlusal relations which are conducive to longer denture service. The present and future problem in the field of prosthesis pertains to the preservation of the denture supporting tissues.

DEATH FOLLOWING EXTRACTION OF TEETH*

By WILLIAM D. NESBIT, Jr., D.D.S., New Haven Conn.

ALTHOUGH the dangers that lie in extraction of teeth are numerous, the dental profession frequently regards the removal of teeth as a trivial procedure of this branch of our specialty. This attitude is probably the result of the immediate amelioration of the symptoms of the inflammatory processes following removal of infected teeth. There are numerous instances in which extraction is ordered without careful study of the individual case and with no thought as to the particular time or the method of procedure. Consequently, we occasionally hear of untoward sequelae or even death following extraction, and only then does the dental profession become awakened to the dangers attendant on this operation.

REPORT OF CASE

History.—An Italian, aged 43, who was admitted to the hospital, Sept. 16, 1926, complained of swelling and tenderness of the right cheek and of a severe headache of several days' duration. The onset of his illness occurred three weeks previous to admission. At that time, he noticed that the right side of his face was swollen slightly. The swelling continued to increase. One week later (two weeks before his admission to the hospital), the upper right first and second molars were extracted. Procain was the anesthetic. In addition, the areas from which the upper right first and second molar teeth had been extracted were curetted by the dentist. The swelling and pain increased, and the patient had a general reaction following this procedure. Two weeks later, the condition was such as to warrant admission to the hospital. The history previous to his last illness was irrelevant.

Examination.—On admission, the patient was in acute distress from a greatly swollen and infected upper right maxilla. The temperature was 101.6 F.; the pulse, 90, the respiration, 20. The blood pressure was systolic, 140, diastolic, 88. Twenty-four hours after admission to the hospital, the temperature by rectum was 103.2 F.; the pulse, 104; the respiration, 32.

The patient was slightly anemic, and undernourished. There was marked swelling and redness over the right cheek. The upper right first and second molars were absent and the other teeth were in very poor condition. The swelling was over a point approximately opposite the upper right molars, and there was a red mass here which was indurated and of semi-fluctuant consistency, tender to palpation. Bimanual palpation of this mass showed the point of maximum de-

*Read before the New Haven Dental Association, April 4, 1928.

*From the Department of Surgery, Yale University School of Medicine.

formity to be the middle of the right cheek. The patient could with difficulty open his mouth to the extent of about 1 cm. The remainder of the physical examination was essentially negative. The clinical picture was that of an overwhelming infection.

The roentgenograms of the upper right maxilla showed that the first and second molars were absent, and the third molar was apparently very loose. There was a marked area of bone destruction with a very faint outline of alveolar process remaining. Roentgenograms and examination by transillumination of the maxillary antrums were negative. There was no tenderness over the right antrum, and no discharge from the nose. The impression at this time was that the condition was osteomyelitis of the upper right maxilla and cellulitis of the right cheek.

A small sinus opposite the upper right first molar was found, from which pus flowed freely.

Operation and Course.—The sinus was enlarged, and approximately 4 ounces of very foul smelling pus was evacuated. The pus cavity was irrigated and drained. The patient had a severe chill during evacuation of the pus. *Staphylococcus aureus* was cultured from this infected area. The white blood corpuscles at this time were 15,600 per cubic millimeter, 76 per cent being polymorphonuclear leukocytes. Subsequently, the sinus was irrigated and dressed daily, much pus being found at each dressing. On September 27, a roentgenogram of the right maxillary sinus showed cloudiness. A thickening of the tissues of the face was interpreted as the cause of this. Examination of the nose showed

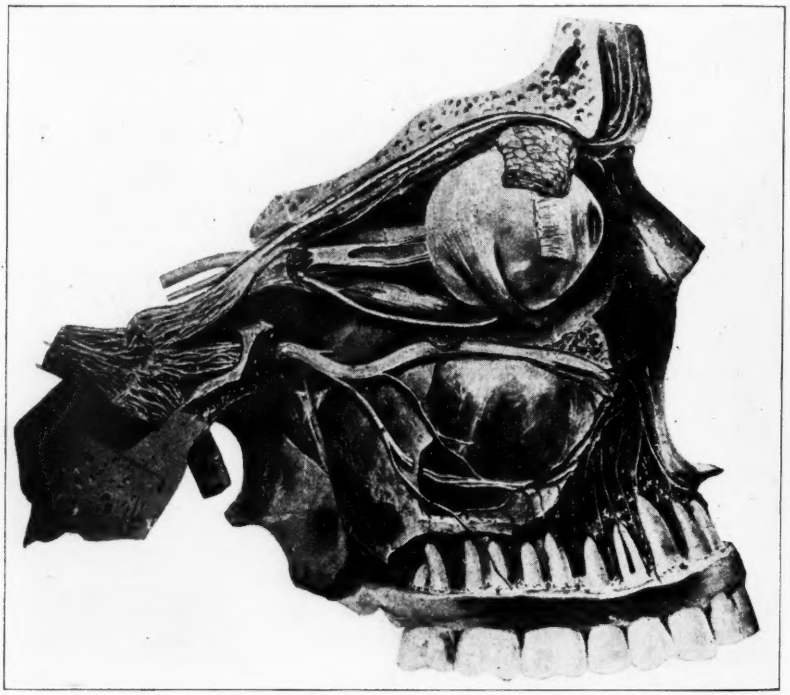

Fig. 1.—Avenue of infection along right maxillary nerve to semilunar ganglion.

no discharge, and there was no tenderness over the right antrum.

September 28, a fluctuant area over the right temporomandibular articulation developed, and this area was incised. About 50 c.c. of thick, yellow pus was evacuated, and a probe inserted encountered rough bone.

September 30, the patient showed signs of meningitis. Kernig's sign and stiff neck were noted. The following day, the spinal fluid was purulent and contained 27,000 cells per cubic millimeter, of which 90 per cent were polymorphonuclear leukocytes. Congestion of the conjunctiva of the left eye developed and protusion of the left eyeball. The motility of the eye was not impaired and the eye grounds were normal. The eye and meningeal symptoms became more marked. Death occurred on the seventeenth day in the hospital, five weeks after the onset of the illness.

Clinical Diagnosis.—Cellulitis of the tissues of the upper jaw, osteomyelitis (right maxilla) and acute meningitis were diagnosed.

Anatomic Diagnosis.—The anatomic diagnosis was as follows: primary: pyorrhea alveolaris; recent operation (extraction of teeth); osteomyelitis of right maxilla, acute maxillary sinusitis; extension of infection along right maxillary nerve to right semilunar ganglion (Figs. 1 and 2), extradural space and tissues about hypophysis and left orbit; fibrinopurulent meningitis; abscess of right temporal lobe with rupture into ventricle (Fig. 3); multiple abscesses of lungs; subsidiary: apical pulmonary scars; pleural adhesions.

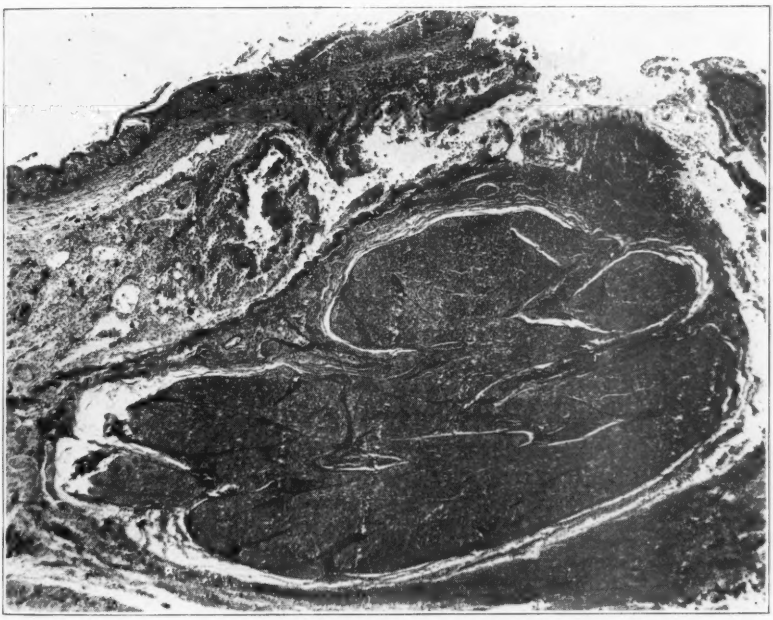

Fig. 2—Photomicrograph of maxillary nerve showing infection (× 24).

Bacteriologic Diagnosis.—Bacteriologic examination revealed *Staphylococcus aureus.*

Necropsy Report.—Body: The body is that of a fairly well developed, but somewhat emaciated, adult white man weighing 58 kg. and measuring 169 cm. in length. Postmortem rigidity is present in the extremities and the muscles of the jaw. The lividity of the dependent portions of the body is extreme. A separated drainage incision 1.5 cm. in length

lies over the right parotid gland region. Elsewhere, the skin is intact, thin and loose. The superficial lymph nodes are palpable only on the right side of the neck. Here, they are discrete, nodular and firm. The neck is greatly increased in size. The extremities show no abnormalities. The head is covered by thick black hair. The scalp is intact. There is no discharge from the ears, mouth or nose. The left eye bulges out. Its conjunctiva is injected and dark red, especially about the pupil. The right eye projects but a short distance above the normal. It is very difficult to open the jaws. The teeth present in the upper and lower jaws are discolored, many are carious, and, by pressure of the gum margin, little beads of purulent material can be expelled. In the right upper jaw, as far as can be seen, the first two molar teeth are absent, and a large, gaping wound present in the jaw is covered by a necrotic clot and purulent material. The tissues about are dark red, soft and fluctuant. An attempt to find the opening into the max-

Fig. 3.—Gross specimen of brain showing abscess of right temporal lobe.

illary sinus on the right side which runs through the right nares is unsuccessful. The thorax is well developed. The abdomen is sunken. The usual incision is made. There is no free fluid in the peritoneal cavity. The surfaces are everywhere smooth and shining. The abdominal viscera are in their usual position. The liver extends to the tip of the xiphoid and to the right costal margin. The entire intestinal tract is moderately distended with gas. The diaphragm is inserted at the fifth rib on either side. Countless adhesions bind the pleura at the apices and portions of the lateral lobe. There is no free fluid in the pleural cavity. The pericardial sac, which is not increased in size, contains an excess of amber colored fluid, which whitens the pericardial surfaces. The pulmonary artery, which is opened *in situ*, is filled partly with a postmortem clot. A small piece of thymus tissue is surrounded by the mediastinal fat.

Heart: The heart weighs 280 gm. There is very little subepicardial fat. The coronary vessels are straight and collapsed. The calibre of the cavities of the heart is not increased in volume. Both the myocardium and the endocardium are intact. The various valve leaflets and cusps appear normal. The openings of the coronary arteries are patent. The coronaries are open and their walls are thin and elastic. Here and there are small, subintimal yellow plaques. The myocardium is red-brown. No fibrous tissue is seen.

Lungs: The lungs are voluminous and moderately heavy. The left lung weighs 650 gm., the right lung, 710 gm. Fibrous tags overlie the apical scars, which are small. The upper lobes on either side crepitate everywhere. In the lower lobe, a number of nodular areas are felt. As the lungs are lifted out in the initial examination of the body, small and large abscess-like areas are seen, having a pale yellow center and an adjacent zone, quite red and gradually shading into the lung tissue, where the red zone becomes darker. As these are cut into, they are seen to extend to the lung tissue, and a thick, creamy pus pours out. This pus contains organisms which resemble staphylococci, and the culture seen shows *Staphylococcus aureus*. Six of these nodules are seen at the right lung and five in the left lung, varying in size from 3 mm. to 2.5 cm. They are more prominent at the periphery of the lung. The pleural surfaces are covered over with flaky, yellow-white, friable fibrin. As this is pulled off, the pleural surfaces lose their luster. On section, the lung tissue appears to be vascular. The bronchi and blood vessels are open and no gross changes are seen.

Spleen: The spleen is large and soft. Its capsule is smooth and stretched. It is slate-gray and so friable that parts of it are removed and it is broken into as the organ is being removed. It weighs 230 gm. and measures 13 by 8 by 4.5 cm. The tissue is somewhat necrotic. The malpighian corpuscles are not distinct. The duct of Wirsung is opened *in situ*. It opens alongside the common duct through the ampulla of Vater.

Pancreas: The pancreas weighs 105 gm. It is of the usual consistency and structure.

Stomach and duodenum: The stomach is distended with gas and black-green semi-solid material. Its mucosa shows the evidence of postmortem necrosis. The mucosa of the duodenum is bile-stained and is intact.

Liver: The liver is congested. It weighs 1800 gm. and measures 33 by 26 by 15 cm. The lobulations are indistinct. Glisson's capsule is smooth and shining.

Gallbladder: The gallbladder is thin and collapsed. Its wall is thin and contains several cubic centimeters of green-black bile. The bile ducts appear normal.

Suprarenals: The suprarenals together weigh 15 gm. Dissection demonstrates the usual structure.

Kidneys: The kidneys resemble each other closely. The left kidney weighs 130 gm.; the right kidney, 150 gm. They measure approximately 12 by 8 by 5 cm. The capsules strip easily. The tissue cuts readily and is congested. The cortex averages from 6 to 7 cm. in depth. The glomeruli are distinct, as are the striations. The pelvis and the medullary portions appear normal. The ureters are normal in course and structure.

Pelvic organs: The bladder, prostrate, seminal vesicles, testicles, epididymis and vas deferens are examined and found to be unchanged.

Intestines: On opening the intestinal tract, no gross changes are found.

Neck organs: The tissue about the tonsils, especially on the right side, is edematous but no purulent material is seen. On section, the tongue appears normal. The mucosa of the larynx and trachea is red-pink and is covered with a tenacious, mucoid material. The esophagus, thyroid and vessels of the neck appear unchanged.

Bone marrow: The bone marrow appears normal on removal of a small portion.

Calvarium, brain and spinal cord: The calvarium is thick. The dura is only lightly adherent to the inner table of the skull. The dura is tense, and as it is cut along the sawline, the brain herniates somewhat. The dura is not dissected from the falx cerebri but is reflected back. There is an increase in fluid in the subarachnoid spaces dipping down in the sulci, but there is no evidence of an inflammatory exudate over the surface of the brain. As the vessel lobes are pulled back, free pus is found along the course of the olfactory bulbs. The left temporal lobe is pulled back readily, but the right temporal lobe is adherent to the dura of the middle fossa, and as it is pulled away, thick, creamy pus pours out from the tip of the right temporal lobe. The basal cysternae and the tissues about the optic chiasm are surrounded by a similar creamy pus. The tentorium cerebelli is then cut. On the cerebellum, there is a thick coating of purulent material, which also fills up the subarachnoid spaces, the basal cysternae and the subdural and subarachnoid spaces about the upper cervical spinal cord. The brain is then removed. A careful search of the cranial venous sinuses shows no evidence of thrombi. Fluid blood fills the majority of the sinuses. Especial attention is paid to the cavernous sinuses on both sides. They are empty. The dura is then pulled away. It is especially adherent in the middle fossa on the right side. The foramen rotundum on the right, through which the maxillary division of the fifth nerve has an exit, is filled with purulent material. This material appears to come along the sheaths of the maxillary nerve and surrounds the gasserian ganglion, bulges out and lifts up the dura about the clinoid processes and extends along the left optic nerve and about the orbital tissue on the left side, producing small pockets of pus, especially along the course of the nerve. There is considerable edema of the tissues of the left orbit. The right orbital tissues are examined but no gross changes are found. The sphenoidal and ethmoidal air cells are open. Their lining mucosa is coated with a shining mucoid substance. A smear from this material shows an abundance of polymorphonuclear leukocytes and gram-negative cocci, similar to staphylococci. The maxillary sinus on the right side is approached from two directions, the dissection being first continued through the air cells until a point is reached where the maxillary sinus is easily opened. As its wall is pushed in from the medial side, a purulent material exudes. The next approach is through the inner part of the floor of the middle fossa on the right side. Again, a purulent material exudes from the maxillary sinus and from the tissues of the soft part of the right cheek surrounding the maxillary sinus. That part of the maxilla from which the first two molar teeth have been removed is split with a chisel. The cortical portion of the bone is soft, friable, vascular, and a deep red. Next, the spinal cord is removed by splitting through the vertebral bodies on either side. There is no fluid in the extradural space. The dura is intact. It is opened *in situ*, and the subarachnoid space, especially along the dorsum, is bulged out with a yellow-white, creamy material. This is cultured and smeared. *Staphylococcus aureus* is obtained from the culture. Organisms resembling staphylococci are seen in the smear along with an abundance of polymorphonuclear leukocytes. There is no apparent exuding of the purulent material along the course of the spinal nerve.

Microscopic Examination. — Heart: The sections of the myocardium show no unusual changes.

Lungs: Sections through the abscesses noted in the gross consist of central masses of purulent material mixed up with blue-staining bacterial or necrotic material, absence of the parenchymal tissue of the lung, a dense infiltration of polymorphonuclear leukocytes and red blood cells. The hole is surrounded by the intact lung tissue, in which there is an extreme distention of the blood vessels with red blood cells. One section demonstrates apical pulmonary scars. There is no evidence of activity.

Spleen: Injection of the sinusoids is noted.

Stomach: The gastric mucosa is intact. Moderate congestion of the capillaries of the lining epithelium is present.

Pancreas: The islands of Langerhans are distinct. The acinar tissue appears normal.

Liver: The section of the liver presents no unusual changes.

Gallbladder: The section of the gallbladder presents no unusual changes.

Suprarenals: The cell structure is distinct.

Kidneys: The glomeruli, tubules and interstitial tissue appear normal.

Pelvic organs: Sections through the bladder, prostate, seminal vesicles, vas deferens, testes and epididymides present the normal structure.

Neck organs: The tonsils are small. There is no evidence of old or recent inflammation of the tonsillar tissues. Sections through the larynx, esophagus and thyroid appear normal. Sections through the lacerated gum in the right upper jaw show a superficial area of necrosis, with an acute inflammation of the gums manifested by the polymorphonuclear leukocytes. There is distention of the blood vessels and lymphatics in these areas.

Intestines: No histologic changes are noted in the sections of the intestines.

Blood vessels: There are slight subintimal thickenings in the sections through the aorta.

Cranial cavity: The brain, meninges, nerves and blood vessels are examined. (Fig. 3.) Sections through the abscess of the right temporal lobe show recent necrosis, no attempt at formation of the wall about the abscess and the presence of a large number of phagocytic cells of the "gitterzellen" type, which are actively phagocytic. No occluded blood vessels are seen near the abscess. The network of fibrin, polymorphonuclear leukocytes and red blood cells coats over the meninges on the right side. The sections taken through the gasserian ganglion and branches show that there is an acute inflammatory reaction involving this ganglion. It has apparently extended from the maxillary tissue along the sheath of the maxillary nerve and the sheath is distended with an acute inflammatory reaction. A similar reaction involves the tissues about the left orbit and about the hypophysis. There is no microscopic evidence of involvement of the cavernous sinus on either side.

Conclusion.—The sequence of events as traced at necropsy was essentially as follows: osteomyelitis of the upper right maxilla; acute maxillary sinusitis; extension of infection along the right maxillary nerve to the right semilunar ganglion, with resulting fibrinopurulent *Staphylococcus aureus* meningitis and abscess of the right temporal lobe of the cerebrum.

DIET ANALYSIS OF ONE HUNDRED AND ONE PERIODONTOCLASIA CASES*

By BENJAMIN TISHLER, D.M.D., Boston, Mass.

THE following report is an analysis of diet lists of a group of periodontoclasia cases taken from my practice, with a few from the clinic of the Harvard Dental School. The purpose of the analysis was to ascertain, with the limited material at hand, any facts which might be of interest. Each patient was requested to record carefully the amount of all food and drink consumed for an entire week, whether at or between meals, and such extra items as candy, cakes and sodas consumed between these periods. As each list was presented, doubtful items were checked up in the presence of the patient and any errors corrected. As a consequence, many lists were eliminated as unsatisfactory, and the final analysis was made from 101, gathered over a period of two years, representing food eaten at all seasons, so that the entire list comprised more than 2,100 meals. The patients' ages varied from 18 to 63 years, and the lesions present from an incipient to a markedly advanced type of periodontoclasia, with or without visible pus discharge.

It should be understood that while the work has been closely carried out as regards the analysis, the amounts of the

*Read before the American Academy of Periodontology, Oct. 21, 1927, as a portion of the Report of the Committee on Scientific Investigation.

TABLE I.—LIST OF FOODS AND THEIR VALUES*

FOOD MATERIAL	WHOLE PORTION	ONE-HALF PORTION	ONE-FOURTH PORTION
A. Meat, including lamb, veal, poultry and pork with the exception of bacon	Amount served at dinner: protein, 20 gm. (calories, 200 to 400)	Amount served at luncheon, breakfast or supper, or at dinner as ingredient of meat pie, on toast, or other meat dish.	Amount served at breakfast, luncheon or supper as ingredient of hash, chicken on toast or other meat dish
B. Fish, including sea foods	Protein, 20 grams (calories, 100 to 200)	See A	See A
C. Bacon or salt pork	Amount served as chief protein dish at any meal: protein, 4 gm. (calories, 100)	Amount served with eggs, liver or other food	
D. Milk	1 glass: protein, 7 gm. (calories, 150)	Milk on cereal or as prominent ingredient of such dishes as milk toast and milk soup	As one of many ingredients, as in custard or creamed vegetables
E. Eggs†	1 egg: protein, 6 gm. (calories, 75)	As prominent ingredient (example, creamed eggs)	Nonprominent ingredient, as of sponge cake or spinach with egg
F. Cheese	Chief protein dish at any meal, such as cottage cheese for luncheon: protein, 12 gm. (calories, 200)	In cheese sandwich	As used in macaroni and cheese or served with pie
G. Whole grain cereal foods	See H	See H	See H
H. Refined cereal products	As breakfast food or any form of rice or macaroni with meat or as ingredient of bread (amount unspecified), cake or two-crust pie; crackers when served with milk, as chief dish of meal: protein, 3 gm. (calories, 100)	One slice of bread, or as ingredient of one-crust pie or of crackers served with soup	
I. Potatoes (Irish)	1, medium-sized: protein, 3 gm. (calories, 100)	As ingredients of hash or other meat dish	
J. Dried beans	As chief protein dish of dinner: protein, 12 gm. (calories, 200)	As chief protein dish at lunch	As ingredients of soup or other meat dish
K. Fresh shelled legumes	As side dish with meat: protein, 5 gm. (calories, 100)	As ingredient of salads, etc.	

*Arranged by Caroline L. Hunt, associate specialist in food and nutrition, Bureau of Home Economics, Department of Agriculture, Washington, D. C., for the author.

†Consider scrambled eggs or omelet a double portion.

TABLE 1.—*(Continued)*

FOOD MATERIAL	WHOLE PORTION	ONE-HALF PORTION	ONE-FOURTH PORTION
L. Spinach	As side dish with meat: protein, 1 gm. (calories, 25)	As ingredient of soup, etc.	
M. Salad vegetables	Whenever appearing on the bills of fare: protein, 1 gm. (calories, 10)		
N. Tomatoes	As salad or side dish with meat: protein, 1 gm. (calories, 25)	As ingredient	
O. Vegetables other than those under K, L, M and N	As side dish with meat: protein, 2 gm. (calories, 50)	As ingredient of salad or any such dishes as carrots and peas	
P. Citrus fruits	1 orange or ½ grape fruit: protein, 1 gm. (calories, 80)	As ingredient of salads, desserts, etc.	
Q. Other fresh fruits	As served uncooked for breakfast or dessert: protein, 1 gm. (calories, 80)	As ingredient of cooked dishes, such as apple sauce, pies and salads	
R. Dried fruits	Same as Q	See Q	See Q
S. Butter	When served with an unspecified amount of bread, 75 calories	When served with one slice of bread or with one helping of cooked vegetables	
T. Other fats than butter, including oil and lard	As ingredient of two-crust pie or rich pastry or salad, 100 calories	As ingredient of one-crust pie	
U. Cream‡	On breakfast cereal or dessert, 100 calories	In tea or coffee or minor ingredient as whipped cream on pudding	
V. Sugar**	In coffee, 50 calories		

‡ Ice cream provides a double portion.

**As ingredient of cake or pie, 2 portions; candy, any amount, 4 portions.

various nutrients on which it was based were computed from a standard devised by the analyst, from the portions of food and drink furnished by the patient. Accurate amounts could only have been obtained by weighing all solids and measuring all liquids before eating or drinking and the same process repeated with such as may have remained unconsumed, the difference having been the net amount actually ingested. This, of course, could not have been done under the circumstances. However, because of the interest shown by the patients and the care taken by them in recording the various items, it is felt that some interesting data have been obtained.

The computation and analysis was made by Miss Caroline L. Hunt, associate specialist in food and nutrition of the

TABLE 2.—STANDARDS USED BY C. L. HUNT FOR JUDGING BILLS OF FARE SUBMITTED BY AUTHOR

REQUIRED NUTRIENTS	FOR A MAN	FOR A WOMAN
Protein	At least 75 gm.	At least 60 gm.
Protein, efficient (of animal foods, peanuts and soy beans)	At least 45 gm.	At least 40 gm.
Calcium	0.67 gm.	0.6 gm.
	(Judged chiefly by amount of milk and cheese)	
Iron	15 mg.	12 mg.
	(Judged chiefly by proportion of meat, eggs, vegetables, fruits and whole-grain cereals)	
Vitamin A	(This is considered adequate if from 300 to 400 calories are obtained from milk, eggs, cream or green-leaf vegetables, and butter is regularly used)	
Vitamin B	(Adequate if from 1,000 to 1,200 calories are provided by other foods than fats, sweets and refined cereals)	
Vitamin C	(Adequate if citrus fruits, tomatoes or raw green-leaf vegetables are regularly used; only alternative, abundant use of potatoes or of fresh fruits in general)	
Roughage	(At least 500 calories should be provided by whole-grain cereals, vegetables and fruits)	
Fuel value	3,000 calories	2,500 calories

TABLE 3.—FORM USED SHOWING FINAL ANALYSIS OF DIET LISTS

ADEQUACY OF DIETS

Judged from bills of fare for one week (amounts not stated): Report by C. L. Hunt, U. S. Department of Agriculture, for the author.

CASE NO. _____ NAME _____

	REPORT*
Protein (estimated grams)	
Protein, efficient (estimated grams)	
Calcium	
Iron	
Vitamin A	
Vitamin B	
Vitamin C	
Roughage	
Water	

* Significance of signs used: +, adequate; + —, probably adequate; —, low; — —, very low; ?, not reported.

TABLE 4.—A SAMPLE DIET LIST (No. 70) AS SUBMITTED FOR ANALYSIS*

BREAKFAST

1 slice toast, 1 cup coffee with sugar and cream.

DINNER

1 cup tomato bisque, 1 slice roast lamb, 3 tablespoons squash, 1 small sweet potato, 1 stick celery, 1 slice jelly, 2 tablespoons chocolate ice cream, 1 slice cake, 1 cup tea.

SUPPER

2 tablespoons baked beans, 2 tablespoons piccalilli, 1 slice bread and butter, 1 slice apple pie, 1 cup tea, 5 or 6 chocolates.

12 P. M.

2 graham crackers with cheese, 1 cup coffee.

BREAKFAST

1 slice toast, 1 cup coffee.

DINNER

1 cup soup, 1 slice cold lamb with hot gravy, 2 tablespoons spinach, 1 slice squash pie, 1 cup tea.

SUPPER

1 egg (Jnornay cheese dressing on toast), 1 slice pineapple, 2 sugar cookies, 1 cup tea.

1 A. M.

1 jelly and cheese sandwich, 1 cup coffee, ½ pound candy.

BREAKFAST

1 slice toast, 1 cup coffee.

DINNER

Steak (very little), 2 tablespoons fried onions, 1 tablespoon mashed potatoes, lettuce (2 leaves) and tomato (3 slices) salad, French dressing, 2 tablespoons caramel custard.

SUPPER

1 Frankfurter, 2 tablespoons potato salad, 1 slice bread and butter, 2 tablespoons caramel custard with cream, 1 cup coffee.

12 P. M.

1 cup coffee, 1 doughnut, 12 chocolates.

BREAKFAST

1 slice toast, 1 cup coffee.

DINNER

Fricassee of chicken (small portion), 2 tablespoons peas, 1 tablespoon mashed potato, 2 tablespoons Indian pudding with whipped cream.

SUPPER

Crabmeat salad, 3 lettuce leaves, 3 heaping tablespoons crabmeat with mayonnaise, 3 rolls, 1 cup coffee, eclair.

12 P. M.

2 toasted muffins with 1 cup coffee, 1 apple.

BREAKFAST

1 slice toast, 1 cup coffee.

DINNER

1 cup soup, 1 lamb chop, 2 tablespoons creamed cauliflower, 1 tablespoon mashed potatoes, 2 tablespoons pepper relish, 2 tablespoons bread pudding with hard sauce, 1 cup tea, 2 sweet crackers.

SUPPER

2 slices bologna, 1 slice bread with butter, 1 tablespoon ketchup, 1 cup tea, 2 bunches Concord grapes, 1 slice fudge cake, ¼ pound candy.

12 P. M.

1 doughnut, 1 cup coffee.

BREAKFAST

1 slice toast, 1 cup coffee.

DINNER

1 slice fried codfish, 2 tablespoons creamed carrots, 1 tablespoon mashed potatoes, 2 tablespoons piccalilli, 1 saucer of sliced oranges and bananas with whipped cream, 1 slice cake, 1 cup tea.

SUPPER

1 small portion scalloped fish, 2 slices bread and butter, 1 cup tea, 1 small slice squash pie.

12 P. M.

1 doughnut, 1 cup coffee.

BREAKFAST

1 slice toast, 1 cup coffee.

* Note the excess of sweets; patient overweight.

TABLE 4.—*(Continued)*

DINNER

1 cup chicken soup, 1 breaded pork chop with tomato sauce, 1 tablespoon mashed potato, 2 leaves of lettuce with French dressing, 1 large slice lemon meringue pie, 1 cup tea.

SUPPER

2 tablespoons baked beans, 3 baking powder biscuits, 1 cup tea, 2 tablespoons piccalilli, 1 slice apple pie.

12 P. M.

2 graham crackers with cheese, 1 cup coffee. Almost one pound of candy.

The patient (Mrs. F. P. B.) is very fond of pepper relish and piccalilli, drinks from 6 to 8 glasses water a day but never with meals and uses plenty of butter in cooking and serving of vegetables.

TABLE 5.—FINAL ANALYSIS OF DIET LIST NO. 70

FINAL TABULATION OF AN INDIVIDUAL DIET LIST

Adequacy of diets judged from bills of fare for one week (amounts not stated) by C. L. Hunt, U. S. Department of Agriculture

CASE NO. 70 NAME MRS. F. P. B.

	REPORT*
Protein (estimated grams)	—
Protein efficient (estimated grams)	—
Calcium	—
Iron	—
Vitamin A	—
Vitamin B	—
Vitamin C	—
Roughage	—
Water	+–

*Significance of signs used:
+, adequate; +–, probably adequate; —, low; – –, very low; – – –, extremely low; ?, not reported.

Bureau of Home Economics, Department of Agriculture, Washington, D. C., who employed a standard in grams for the various required nutrients in whole, half and quarter portions, both for a man and for a woman. From these, an analysis was prepared for each diet list using the terms, adequate, probably adequate, low and very low as the results indicated. The recorded nutrients were protein, protein efficient, calcium, iron, vitamins A, B and C, roughage and liquids. It was originally intended to include iodin, phosphorus and calories, but as the question of the influence of iodin is still uncertain, it was omitted. Miss Hunt also decided that the data on phosphorus were too insignificant to be included. Calories, on the other hand, are affected by the amount of sugar and fats in the food and also by the amount of butter, oil or syrups used. Because these foods are so concentrated and are used in varying amounts, it seemed use-

TABLE 6.—DIET ANALYSES, AGES EIGHTEEN TO THIRTY YEARS

NAME	No.	Age	Sex	Pro.	Pro. E.	Ca.	Ir.	VITAMINS			Rough-age	Water	Clinical Symptoms
								A.	B.	C.			
L. O.	104	18	F	+	+	—	+	+	+	+	+	- -	General but early
T. L.	13	21	F	+	+	- -	—	- -	+	+	+ -	—	Advanced
D. K.	89	23	M	—	—	—	+-	—	—	+-	—	—	Advanced
F. K.	79	24	F	+	+	+	+	+	+	+	+	—	Early
M. B.	68	24	M	- -	- -	—	—	—	—	—	—	—	Generally advanced, with suppuration
H. A.	78	25	F	- -	—	—	—	—	—	+	+	—	Locally advanced with suppuration
G. C.	48	25	F	—	—	—	—	—	—	+	—	- -	Locally marked
L. K.	71	26	F	+ -	+	—	+	—	+	+	—	—	Moderately advanced, with suppuration
E. G.	31	26	F	—	—	—	—	—	—	—	—	- -	Very advanced, marked suppuration
L. C.	8	26	F	+ -	+ -	- -	+	- -	+ -	+	—	+	Moderately advanced L. S. lateral involved
M. C.	41	27	F	—	—	—	—	—	—	—	—	—	Generally advanced, with suppuration
D. M.	56	28	F	+	+	+ -	—	+ -	+ -	—	—	—	Moderately advanced
M. Q.	24	28	F	—	—	—	—	+	—	+	—	—	Generally advanced, with suppuration
E. C.	111	28	F	+	+	+	+	+	+	+	+	+	Very advanced, with suppuration
J. B.	73	29	M	—	+ -	+	+	—	+	+	+	—	Advanced
M. G.	100	30	F	—	—	—	—	—	—	+	+	- -	Advanced, with suppuration
H. M.	98	30	F	—	—	—	—	—	—	—	—	+	Moderately advanced, with suppuration
A. B.	86	30	M	—	—	—	+ -	—	—	+	+ -	- -	Advanced, with suppuration
E. G.	30	30	F	+ -	+ -	—	+ -	—	+ -	+ -	—	- -	Very advanced, with suppuration

TABLE 7.—DIET ANALYSES, AGES FORTY-NINE TO SIXTY-TWO YEARS

NAME	No.	Age	Sex	Pro.	Pro. E.	Ca.	Ir.	VITAMINS			Rough-age	Water	Clinical Symptoms
								A.	B.	C.			
G. B.	99	62	F	—	—	—	—	—	—	+	+	+	Advanced
E. K.	96	62	F	- -	- -	+	—	+	+	+	+	+	Advanced
E. H.	18	60	F	+	+	—	+	—	+	+	+ -	+	Moderately advanced
C. F.	52	60	F	—	—	— ?	— ?	—	—	—	+	+	Advanced, with suppuration
E. C.	81	59	M	—	—	—	—	—	—	—	+	—	Very advanced, with suppuration
S. K.	113	57	M	+	+	?	+	+	+	+	+	+	Advanced, with suppuration
G. M.	66	57	M	+ -	+	—	+	—	+	—	+	+	Very advanced, with suppuration
A. P.	82	55	M	+	—	+	+	—	+	+	+	—	Advanced
J. G.	61	55	M	+	+	+ -	+	—	+	—	—	+	Advanced
P. O.	40	54	M	+ -	+	—	+ -	—	+ -	+	—	+	Advanced
I. S.	65	53	M	—	—	—	—	+ ?	—	—	—	+	Very advanced, with suppuration
E. D.	109	51	M	+	+	+	+	+	+	+	+	+	Advanced
R. R.	53	52	F	- - -	- - -	—	—	—	—	—	—	- - -	Moderately advanced
E. G.	93	52	F	—	—	- -	—	—	—	+	—	—	Advanced
B. C.	7	50	F	+	+ -	+ -	+	+ -	+	+	+ -	—	Advanced
M. K.	23	50	F	—	—	—	—	—	—	+	—	+	Moderately advanced
J. A.	69	50	M	—	—	- -	—	- -	- -	—	—	- -	Advanced
T. H.	37	50	F	+ -	—	—	+	—	+	—	+	- -	Advanced, with suppuration
F. A.	2	50	M	—	+	- -	—	—	—	+	—	+	Advanced
L. H.	16	49	F	—	—	—	—	+ -	—	—	—	—	Advanced, with suppuration

less to consider them except as they affected the rest of the diet. As an example, when butter, sugar and such dishes were used in large amounts, it was presumed to indicate that the diet was overabundant or deficient in essential nutrients such as protein, minerals and vitamins.

The final computation of the lists disclosed one conclusive fact, i. e., that a very great proportion of the patients, both young and old, ate a poorly balanced diet, in some cases, alarmingly so. Many women gave the information that they were accustomed to restrict the amount and kind of food eaten to avoid an accumulation of surplus flesh, a condition which, as far as could be determined, did not affect the men, whose diet analyses, by the way, disclosed an equally marked deficiency in many cases. In computing the averages of the various elements, it was found that three items of the nine already referred to were conspicuously low: calcium, vitamin A and liquids (mostly water). Of the 101 recorded cases, about 96 per cent showed an unbalanced diet; in other words, ninety-seven individuals were found to have eaten unwisely in one or more of the recorded nutrients. Nine showed a deficiency in every item (a 100 per cent unbalanced diet). Seven were deficient in from five to eight items, with the balance, necessary to complete the nine, recorded as probably adequate only (not an adequate amount in the entire number of nutrients). The balance of the diet lists varied from one to nine adequate items, and the latter group, which represented those who ate a balanced diet, numbered but four (of the original 101 examined).

In order that a definite idea of the diets as a whole may be obtained, two groups of opposite age extremes have been chosen and recorded. One represents lists submitted by younger patients (nineteen in number) whose ages ranged from 18 to 30 years. These showed a general nutrient deficiency, with but one exception, and this diet list was adequate in all items. Three lists were deficient in all nutrients, one in all but two and an-

TABLE 8.—FINAL TABULATION DIET ANALYSES OF ONE HUNDRED AND ONE PERIODONTOCLASIA CASES WITH POCKET FORMATION

	Adequate	Very Adequate	Probably Adequate	Questionable	Low	Very Low	Extremely Low
Protein	40		10		47	3	1
Protein Ef.	46		9		42	3	1
Calcium	20		7	3	62	9	
Iron	45		10	3	43		
Vitamin A.	24		10	4	60	3	
Vitamin B.	51		8		41	1	
Vitamin C.	59	1	6		34	1	
Roughage	43		12		45	1	
Water	31		1	4	52	12	1

other in all but four, all of which were recorded as adequate. The clinical symptoms of both groups varied from an incipient to a markedly advanced type, with the advanced predominating. The other group of twenty, chosen from the opposite age extreme (49 to 63) disclosed a similar general diet deficiency. In fact, the lists of both groups could have been reversed without appreciably affecting the result. The balance of the remainder of the lists, which completed the 101 chosen, i. e., those between 30 and 49 years, showed the same general deficiency and symptoms as the two groups just referred to, which would lead one to believe that, as far as the experiment is concerned, diet is not a conclusive etiologic factor in periodontoclasia when individuals are considered, but when the 101 cases are examined collectively, one cannot overlook the general lack of the calcium and vitamin A intake, and also that of water, which is so important in fecal elimination and bears a direct relationship to intestinal toxemia, long recognized as an etiologic factor in periodontoclasia.

Of the two groups, just reported, it was hoped that the younger would offer the more promising material for consideration because the periodontal disintegration had its inception at a more recent date than in the case of the older groups, but, as has been observed, it offers no conclusive results on which a positive statement can be made. It would seem, therefore, that more definite data could be obtained by an examination of the diets of still younger patients, of high school age or younger, if found necessary. The advantage of this is obvious, because it is apparent that a knowledge of the character of the diet eaten during the formative period of life, when early periodontal lesions have their inception, is most important. It cannot be disputed that many persons make radical changes in their diet as soon as they are permitted to choose it, as in adult life, thus making the task of relating diet to periodontoclasia a difficult one. It is safe to assume that this could be determined with more certainty during childhood or youth, for the closer that the etiology (deficient diet) and the possible effect (periodontoclasia) can be brought together, the more definite should be the findings which will prove their exact relationship.

It is hoped that further work of this kind will be undertaken, because those familiar with dietary problems who have been consulted are of the opinion that such experiments offer promising results. A suggestion is hereby offered to those who may desire to undertake work of this kind that a dietitian be enlisted to supervise the accumulation of the diet lists, because of her expert knowledge in such matters.

OCCLUSODONTIA IN RESTORATIONS*

By E. RAY BROWNSON, D.D.S., Los Angeles, Calif.

THE crux of restorations is balanced physiologic cuspidation; in other words, cuspidation as near the ideal anatomic depth and sharpness as the mechanics of the individual case[1] will permit. In 1917, I established, at the University of Southern California, the first course in restorations, and now after fourteen years of experience[2] with the various technics and instruments, trying to attain an ideal, it is with some enthusiasm that I submit the following finding, namely, that the relator and plastic technic, permitting an unlimited range of adjustment to register curves or planes, are balancing fundamentals for those who know the principles, technical steps[3] and classifications laid down by Joseph Homer, of New York City, who has given us, among other things, the only instrumental incisor guidance that is technically useful.

This finding is based on practice and the opinions of Sam Supplee; W. A. Giffen, of Detroit; John H. Hospers, of Chicago; E. J. Weaver and G. H. Doregkamp, of Milwaukee; Clark Giles, of San Francisco; H. C. Peisch, J. W. Martin and Lee K. Stewart, of Pasadena, and E. Ray Kibler, E. E. Kirtlan, C. M. Benbrook, W. H. Spinks, C. G. Hartley and G. B. Baird, of Los Angeles.

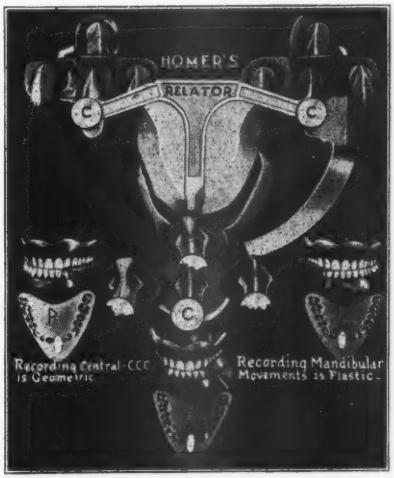

Fig. 1.—Technic of registrations and making negatives of mandibular movements. A detailed description is given on pages 136-139 of THE JOURNAL, January, 1928. Figures 1-12 illustrate Technic 3.

Restoration cases naturally fall into three classes, requiring different technics for their remedy.

Class 1 includes cases in which the occlusion is partly traumatic.

Class 2 includes cases in occlusions that are entirely traumatic.

*"Occlusodontia" is defined on page 141, THE JOURNAL, January, 1928.

*Read before the Section on Partial Denture Prosthesis at the Seventieth Annual Session of the American Dental Association, Minneapolis, Minn., Aug. 22, 1928.

1. Homer, Joseph: Pacific Dent. Gaz., 36:213 (April) 1928.

2. Bull. Col. Dent., Univ. Southern California, 1918, p. 8.

3. Homer, Joseph: Occlusodontia Notes, 1927.

Class 3 includes cases in which orthodontic treatment or bite-advancement is indicated.

All three technics include reproducing mandibular movements and the actual relation of the maxillary line of occlusion with the mandibular line of occlusion or curve of spee under function

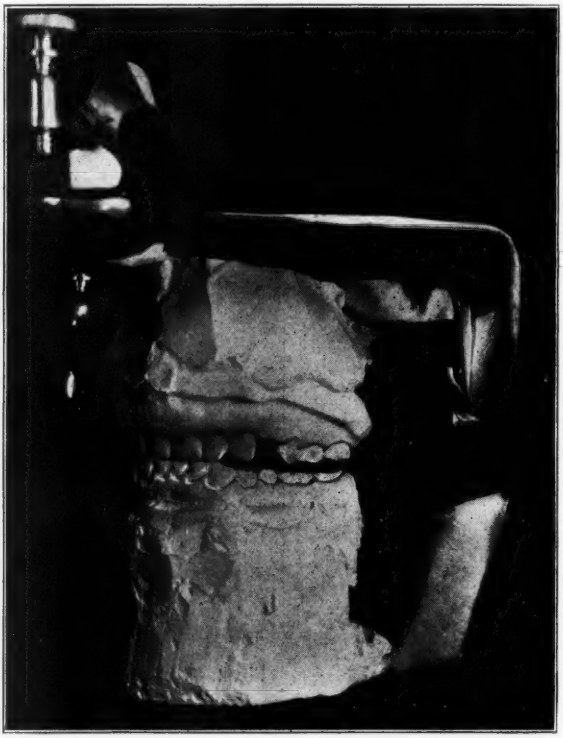

Fig. 2.—Survey for lack of balance, etc.

(Fig. 1) for a preliminary survey (Fig. 2) and removal of trauma when present.

Technic 1 is used principally where many teeth are to be supplied in both jaws. The occluding abutments or inlays which collectively establish cusp height and angulation are made in the usual way and registrations are taken before the impressions. The case is mounted on a relator, and after the instrumental condyle and incisor paths or negatives are made, the cusps of the abutments and supplies, or the models of the opposing natural teeth, are lengthened for balance, which is "equalized pressure on the supporting tissues," and shortened for interference. The natural teeth whose models are so altered must be correspondingly altered after construction is complete. This is accomplished by placing the restorations

in the mouth, then spot grinding and placing inlays where necessary, having the patient grind balancing angulations on the wax forms for these inlays in the mouth. Finished construction is finally adjusted and milled on the relator and in the mouth.

lish cusp angulations and height (having regard for central occlusion only). Castings are then made for all other preparations to just cover the cavities with no cuspidation.

Registrations and impressions should be taken with the completed inlays in

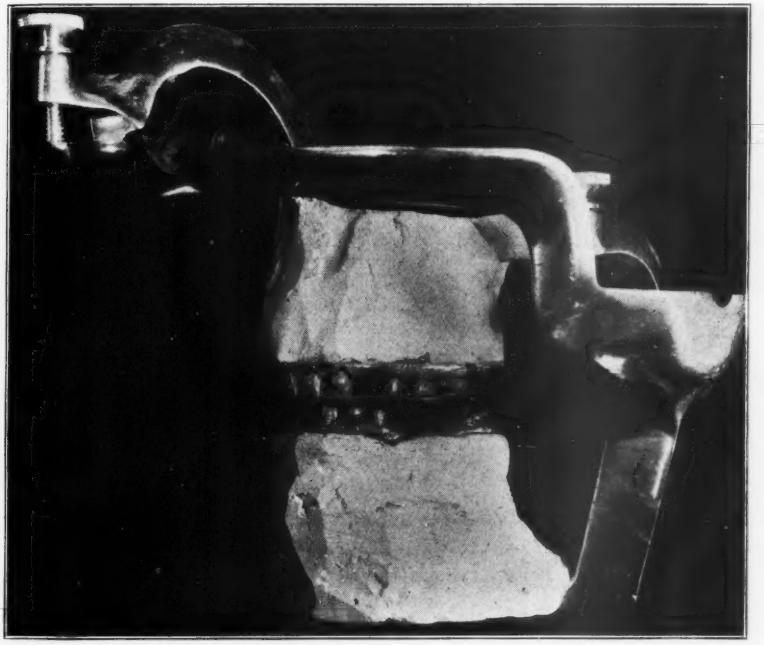

Fig. 3.—Bite opening and advancing splint.

Technic 2, which I helped to develop, is used for bite-opening cases having all natural bicuspids or second bicuspids and first molars retained, and is principally used where no teeth are to be supplied and the entire occlusion is constructed. After all preparations are completed, castings are formed on the four bicuspids or second bicuspids and first molars to open the bite and establish the bicuspids or "molars" and the castings without cusps in the rest of the teeth. Casts are mounted on a relator, negatives are made and wax is built up to balance (having a regard for anatomy) on all cuspless castings, and a second set of castings is made direct to the first castings. Any lack of balance or interference is corrected in the bicuspid or "molars" castings, after which

the case is finally adjusted and milled on the relator.

Technic 3 is indicated where orthodontic treatment[4] or bite advancement is advisable, and may be used on all bite-opening cases. A bite-opening splint establishing counters of cusp height and angulation is made of vulcanite (Fig. 3). Any bite advancement is also recorded by this splint, and after adjustments, registrations are taken of the masticating positions with it in the mouth and reproduced on the relator. This splint is then removed from the models and wax cusps are built on either upper or lower cusps of the models mounted on the instrument, wherever necessary to give balance and esthetics. All upper wax cusps are joined together and all lowers likewise, and are cast in one piece, or separately and assembled, such clasps being made as are necessary for retention. This makes two more, called balancing splints (Fig. 4), which are adjusted on the instrument and in the mouth by spot grinding, milling and cusp lengthening, after which a lock incisor splint (Fig. 5) or fourth and last is cast, the wax being formed with the balancing splints in the mouth.

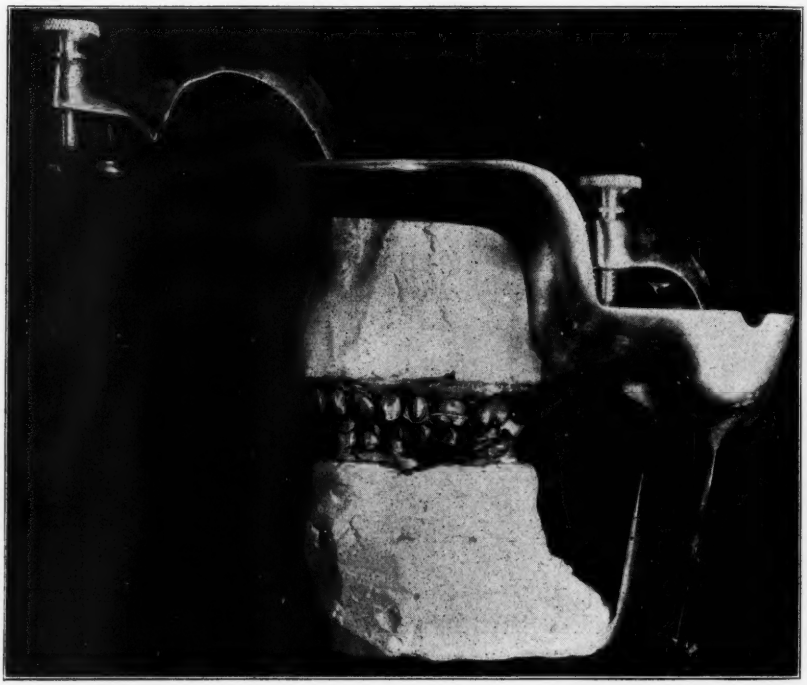

Fig. 4.—Gold balancing splints.

The lock incisor splint (to maintain and check correct central occlusion in the

4. Footnote 1, p. 214.

open bite relation) and one balancing splint are placed in the mouth, and the operator proceeds with construction (Fig. 6) opposite the balancing splint, which is then removed, and construction finished (Fig. 7) on its side, the lock incisor splint being retained in position till all construction is complete.

1. A double compound impression is best for surveys, or occlusal surfaces. An impression is seated, removed, and drawn places trimmed, semi-cooled, reseated and removed. Then, a small roll of soft compound is placed in the thoroughly chilled impression, so placed and seated as not to enter undercuts.

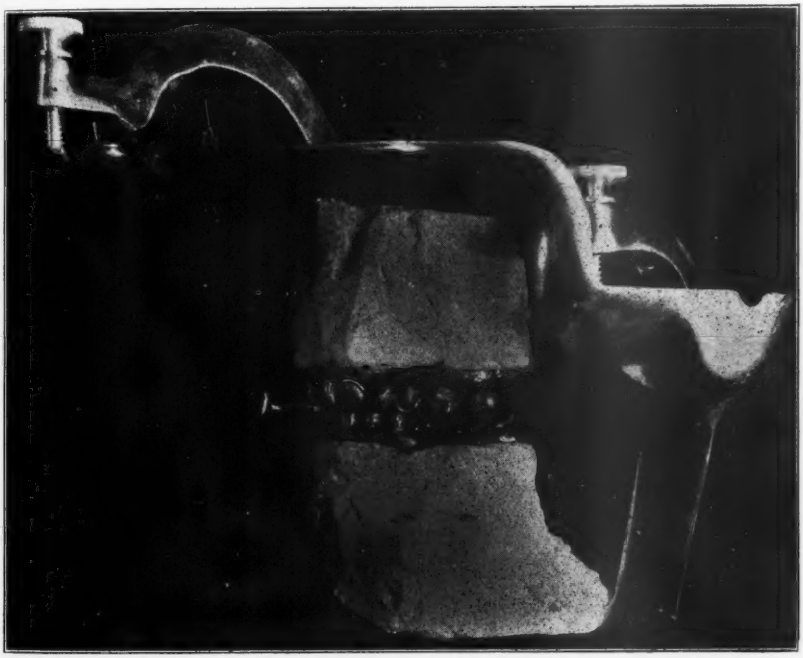

Fig. 5.—Upper gold balancing splint and incisor lock splint (gold) ready for lower construction.

All construction is made with regard to central occlusion only as all balancing cusp angulations have been established by the balancing splints.

The finished construction should be adjusted and milled on the relator and finally in the mouth to overcome coefficient and cumulative error.

Pertinent findings by Dr. Homer are: Reheating this surface is only permissible by dipping in hot water for five seconds.

2. The occlusion is guided horizontally by the condyle paths and vertically by cusps. If the occlusion were guided solely by the condyle paths, there would be no cusps. If it were controlled solely by cusps (which would include maxillomandibular develop-

ment), there would be no traumatic occlusion.

3. Registration of lateral and protrusive mandibular stresses is artistic and plastic rather than mechanical or geometric, as follows: Trying to measure tissue resiliency in pounds or controlling it with a spring mechanism is of no practical value as a few pounds compress the tissues as much as many for all practical purposes. So far, the best technic is to have patients close gently on a relator register between cusp inclinations selected for the case, instructing them to feel the teeth touch through the wax (which in reality is usually not quite or barely through at one or two places). Then the wax is chilled and the patient is asked to close again hard in the same position to correct distortion. This frequently necessitates vulcanizing temporary registration plates (refitted to cast with a compound lining) and removing obvious trauma before registrations, which should always be checked.

4. Two registrations should be taken for each masticating position: one, end to end, and another one one-sixteenth inch beyond. This permits more accurate reproduction of condyle and incisor path curves on the relator.

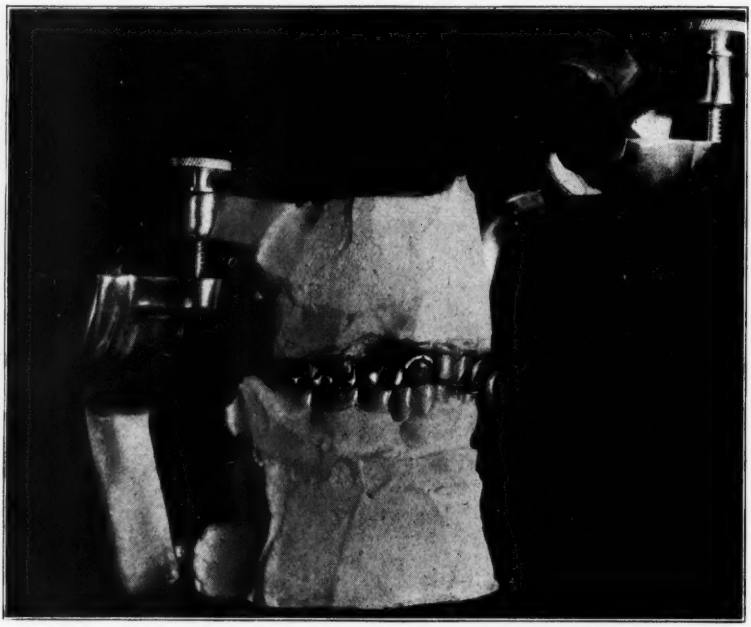

Fig. 6.—Lower construction complete, with upper balancing and lock splints.

5. Central occlusion is best obtained by having the patient let the jaw hang loosely about one-half inch from the maxilla and fall toward the maxilla on

a horseshoe-shaped piece of softened true wax. After chilling, say to patient "Let the jaw hang." "Let your teeth fall together," and "Now, bite hard." Repeat until the patient gives successive identical closures from the same relaxed position.

6. Face-bows and gothic arch tracings are time-wasting complications because better work cannot be effected by their use.[5]

7. Opening the bite 2 mm. usually removes all occlusal trauma. Starting points are 15, 18 and 21 mm. (for short, medium and long crowns) between the gingival enamel margins of opposing central incisors.

8. Balance is direct or indirect; that is there may be contact on each tooth, or there can be from one to three contacts that will balance masticating forces on a number of opposed, joined teeth.

Fig. 7.—Upper and lower construction complete, with lock splint.

9. Balancing restorations where the bite is opened is not for the average dentist or pocketbook.

10. Natural dentures of a healthy patient should not be mutilated by balancing restorations.

5. Homer, Joseph: Disc. of Hanan, R. L.: Bull. Chicago Dent. Soc., 6:8 (Aug. 6) 1928.

DISCUSSION

J. H. Hospers, Chicago, Ill.: Dr. Brownson failed to stress the use of the instrument

with which this difficult task was accomplished. There should be no stint of praise to Dr. Homer for giving us this unique and logical method of reproducing the movements of the human jaw. Occlusodontia[6] or the science and art of relating the occlusal surfaces of teeth in the proper functioning plane has received an epochal contribution in the relator, which is capable of making an individual negative of the condyle path and incisor path for every patient. There is nothing geometric about it, and its use requires only a common sense interpretation of Nature's laws. I have used a relator for three and one-half years and find it perfect. In fact, any imperfections in the balance of a completed denture can readily be traced to an error on my part. When it is necessary to open a bite, I have found the following a satisfactory method of obtaining the condylar registrations: Use the flat horseshoe plate as a tray and take an impression of the upper one-third of the lower teeth. Apply wax and lampblack for the making of the gothic arch. Apply vaseline to the upper surface of the horseshoe plate and replace over the lower teeth. Soften the modeling compound and form into a roll, place on top of the horseshoe plate and close the upper teeth into it. Fasten the Gysi incisor path marker into the compound at the median line and cool thoroughly. The compound impression of the upper teeth will slide freely over the upper surface of the horseshoe plate. Drop the pin in the incisor path marker and develop the triangle. Then, with the pin, in the apex of the triangle, make lock blocks of compound to hold the parts together. Remove everything from the mouth and lock the horseshoe plate and upper compound together. Fit the models into the compound and mount on the relator. Loosen the lock

Fig. 8.—Completed case in working position.

6. Defined on page 141 of THE JOURNAL, January, 1928.

blocks and raise all screws on the relator. Fill the cups with soft compound and make all movements by following the gothic arch tracing. The entire range has been reproduced without checkbites and before we have any cusp inclinations to help guide us. It is then possible to articulate the restorations almost perfectly. The balance can then be refined by using the usual technic of wax registers. The detail will be simplified to a large extent in Technic 2 if this procedure is followed. "Duration of denture service depends on the condition of the supporting tissues. Tissues in turn are affected by the dentures which they support. Conservation of these tissues is the paramount problem. The solution is balanced occlusion."[7] I do not think that deep cusps are so essential as continuous contact of the upper and lower facets in all working positions. Food is cut and chopped through the scissors action and not through milling as of long cusps working in deep sulci. The relator makes possible the production of this continuous contact and that is why such efficient restorations can be produced. I do not like the idea of restoring a mouth with large quantities of gold. Instead of gold crowns, porcelain veneer crowns and occlusal pin

Fig. 9.—Completed case in balancing position.

7. Campbell, D. D.: Disc. of Wright, W. H.: Anatomic. Influences on Establishment of Balanced Jaw Relations and Balanced Occlusions, J. A. D. A., 15:1102 (June) 1928.

onlays held in place with three or four pins are practicable. Porcelain jacket crowns must be watched and checked frequently for stress. They do not wear down and, in time, all the stress of closure is taken by them. Unless occasional corrections are made, overstress will cause breakage or serious impairment to the supporting tissues. Generally speaking, radical opening of the bite is not attended with the most nearly permanent results. The anterior teeth should be completed first to get the proper length and contour. Modeling compound impressions are the best; but one must know how to take a modeling compound impression. I agree with Dr. Homer's findings on the futility of the face-bow; but the use of the gothic arch tracing has been a valuable aid and time saver to me.

Dr. Brownson (closing): My paper was given as a technic, describing what has proved best in my hands after many years of trying different technics and procedures. Therefore, I did not go into detailed discussion of the relator. I am glad Dr. Hospers did this, but he cannot mean that the relator is not geometric because it is used technically and accurately. In order to take accurate checkbites or registrations, we must first establish cusp angulation and height, so as to record accurately in the register any discrepancies in the horizontal relations of the maxilla to the mandible in function, which interests us more than the movements of the condyle. Taking registrations is not accomplished by sliding the teeth. The mouth is opened and closed in the position in which we wish to take the registration. Refer to the third pertinent findings by Dr. Homer, in my paper. I am sorry to see Dr. Hospers going to the extra trouble of making gothic arches. If we look in the incisor guide com-

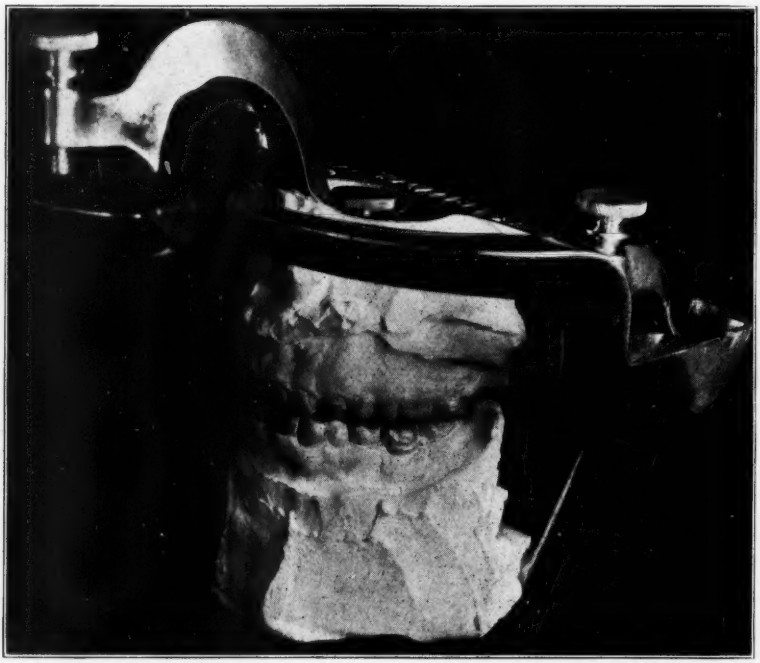

Fig. 10.—Completed case in anterior (protrusive) position.

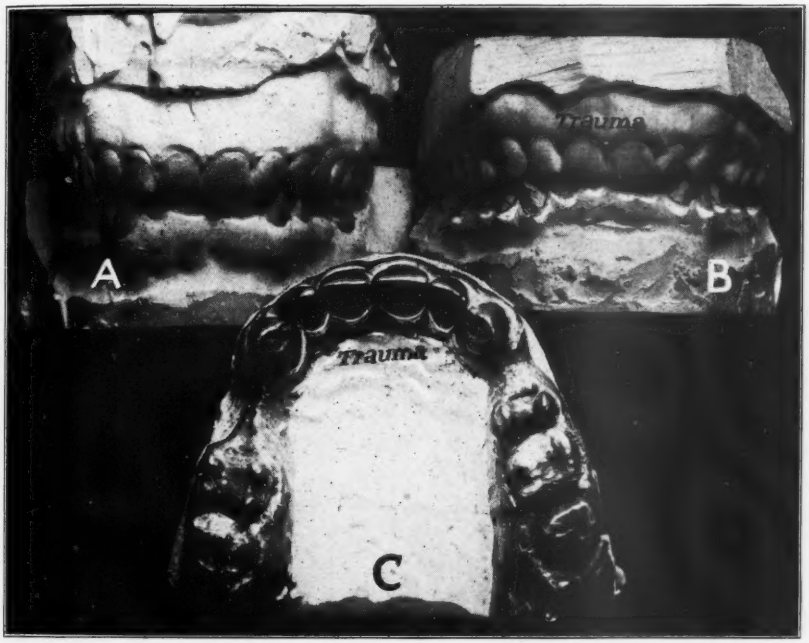

Fig. 11.—*A:* Completed case with a 5 mm. bite opening and a 3 mm. advance. (The bite was advanced to eliminate orthodontic movement of the lower six anterior teeth, this being preferred by the patient.) *B:* Case as presented and hypertrophy of the anterior gums due to trauma. *C:* Lingual gum hypertrophy due to trauma.

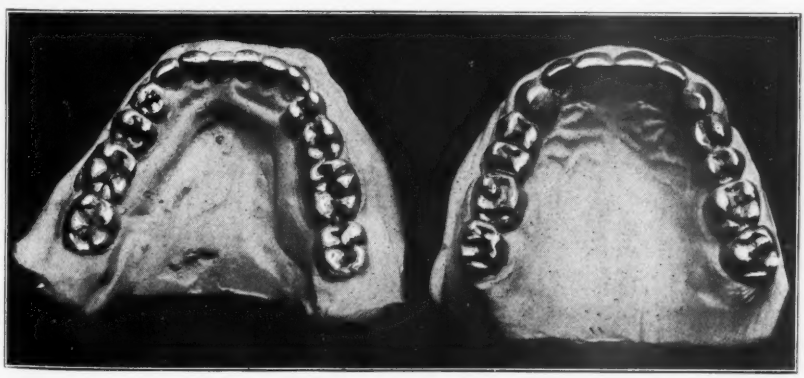

Fig. 12.—Construction anatomy.

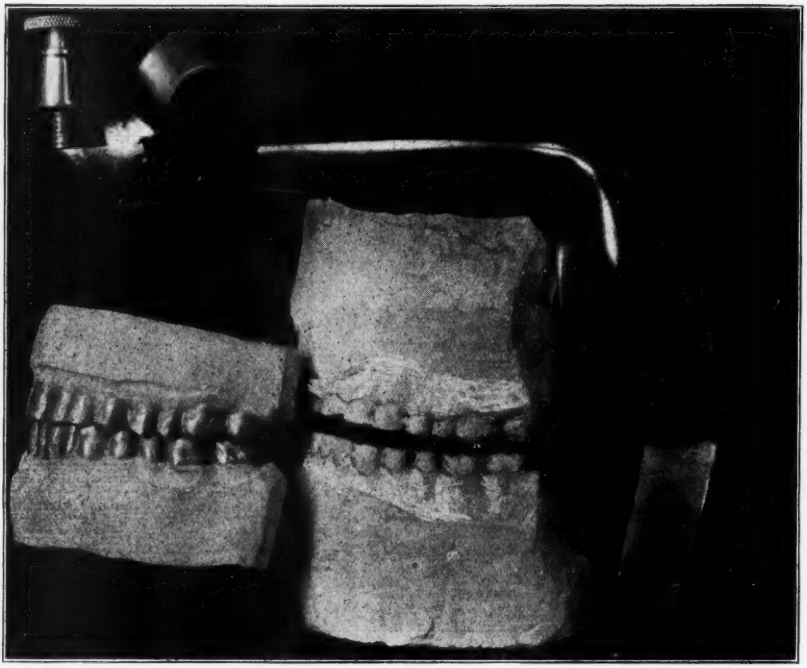

Fig. 13.—Bite opening of 7 mm. which retruded the lower jaw 2 mm. in central relation as compared to central relation before construction. Casts at the right show the case as presented. (Technic 2.)

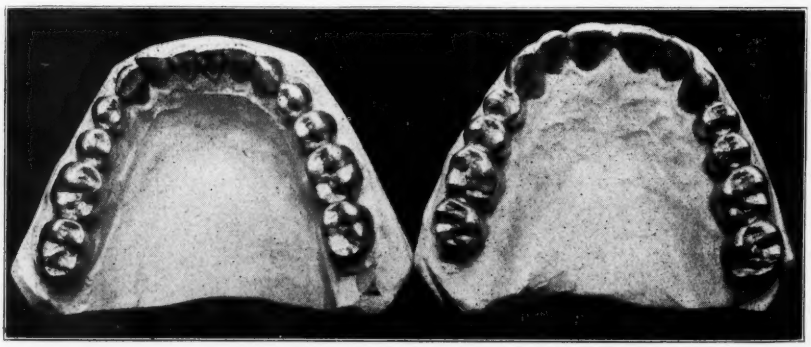

Fig. 14.—Anatomy of completed construction. (Technic 2.)

Fig. 15.—Case before and after construction; a reduction of the abnormal relations of the lateral cuspid and bicuspid. (Technic 1.)

pound after the negative has been made, we will see the useful part of the gothic arch, not one made on a flat surface, but a path for the incisor guide, up and down, over cusp angulation and height. Therefore, we see why it is time wasted to trace a gothic arch. I have records showing tissues returned to normal and teeth, loosened from trauma, tightened in from seven to ten days, in cases in which normal cusps rather than shallow ones full of facets were used, because, with normal cusps, there is a lack of frictional contact.

USE OF GOLD FOIL IN THE FILLING OF CAVITIES IN THE ANTERIOR TEETH*

By LEROY L. HARTMAN, D.D.S., New York City

THE purpose of this paper is to present a definite stroke technic in cavity preparation and to emphasize certain points in the manipulation of gold foil which will make its use comfortable for the patient and a pleasure to the operator.

In our study and treatment of caries of teeth, our first consideration should be for our patients. The earlier we detect caries and effect its removal, the greater the possibility of safeguarding the vitality of the dental pulp. The cavities should be so prepared as to minimize the recurrence of caries and the filling materials used should restore the teeth to their normal function.

When caries attacks the anterior teeth and the patients present themselves to us for treatment, they are particularly interested in the appearance of any restoration which we might suggest and one of their first questions is, usually, "Is the

*Read before the Section on Operative Dentistry at the Midwinter Clinic of the Chicago Dental Society, Jan. 15, 1929.

filling going to show when I smile or laugh?"

The responsibility is then placed on our shoulders to so plan the restoration of the lost tooth structure that we will safeguard the tooth against the recurrence of caries and at the same time make our fillings blend with the normal lines and shadows on the surfaces being restored.

Before starting any cavity preparation in the anterior teeth, we should first examine carefully the amount of tooth structure involved by the caries, and we will note in proximal cavities that distal cavities are less conspicuous than mesial cavities. Also, certain gingival third cavities, especially in upper and lower cuspids, are hardly noticeable because they are covered by the lips during conversation and, in most cases, even when the patient is laughing.

In order to plan the lines of the cavity margins so that they will present the least visibility, the operator should note several important points. He should first examine the contour and study particularly the areas of shadow on the surfaces to be involved. After making these observations, it would be easy to prepare a proximal cavity in such a way as to reduce its visibility in the mouth by simply making the labial margin as nearly parallel as possible to the proximolabial surface angle approached or involved.

In a gingival third restoration, the visibility is reduced by having the exposed margins parallel the surfaces that they approach; for instance, the incisal wall parallel to the horizontal plane of the tooth, the mesial wall of the cavity parallel to the mesial surface and the distal wall parallel to the distal surface.

Secondly, he should note that all angles on the labial and proximal surfaces of the anterior teeth are gently curved, and it is therefore wise, from an esthetic standpoint, not to leave sharp angles in the enamel walls of cavities but to curve them gracefully.

In the selection of a filling material, the operator is again called on to use judgment. The best looking filling material at the time of insertion will not always be the most harmonious restoration after a short period of time.

From an appearance standpoint, a filling material should retain its contour and a finish which would blend with the rest of the tooth being restored. It also should not affect the vitality of the pulp, it should not change color in the fluids of the mouth nor should it have the tendency to discolor the structures of the tooth in which it is inserted.

In the restoration of lost tooth structures, the original contour should be reestablished, and, in some cases, by slightly exaggerating the contour, the light rays may be so reflected that even large restorations may be made to blend very well in the mouth.

In using a metal filling in the anterior teeth, the visibility is reduced by giving the surface a dull finish rather than a highly polished one. A careful operator should therefore have no difficulty in meeting the requirements of a patient concerning the appearance of restorations in the anterior teeth.

The second question that is usually asked by patients is "How much pain am I going to suffer?"

The operator should realize that he is working on living tissue, and in order to minimize the pain to the patient, he should have a thorough knowledge of the structures with which he is working, the sensitive areas in the teeth and the soft tissues surrounding the teeth, as well as the type of instruments best adapted to prepare cavities painlessly.

An anesthetic is rarely necessary in cavity preparation.

Thermal changes are particularly noted by the dental pulp, and, therefore, in preparing cavities in teeth, it is advisable to use instruments which will perform the required steps with the creation of a minimum amount of heat.

Owing to the size of the cavities and the access, the use of burs and cutting instruments are particularly indicated in the anterior teeth. The efficiency of sharp inverted cone burs and keen cutting instruments has long been emphasized. But there is one other thought which should be stressed, and that is the employment of all instruments with deft strokes, each stroke accomplishing an important step in the cavity preparation.

In order to minimize pain where the soft tissues surrounding teeth are involved: 1. Ligatures should not be snapped through the contact points but just eased through. 2. The rubber dam should be adjusted carefully, the edges should be inverted, and only in the rarest instances should we resort to ligation. 3. When clamps are indicated, they should be adjusted gently and removed just as soon as possible after the filling has been placed. 4. If gold foil is being employed as the filling material, an injured pericemental membrane may be avoided by exerting a sufficient amount of pressure with the plugging or holding instrument before each mallet blow strikes. The hand mallet used by a competent assistant permits the operator to exert a sufficient amount of pressure before each blow and also the rhythm of steady and well directed blows on a plugging instrument has a soothing influence on most patients. 5. The mechanical separator should remain on the teeth just as short a time as possible. It still has a place in separating teeth after filling in order that the contact point may be finished. In the finishing of fillings, knives, files, disks and strips should be used in such a way as to minimize the laceration of the soft tissues. 7. On the completion of the operation, the gums should be thoroughly massaged and washed with a mild antiseptic solution, and the patient should be dismissed.

The third question from the patient is "How much time is going to be required?" A safe estimate on the time necessary to prepare and fill with foil a cavity starting in a defect such as a lingual pit in a lateral would be from fifteen to twenty-five minutes. A small gingival third cavity could be prepared and filled in from twenty to thirty minutes. A larger gingival third restoration could be prepared and filled in from thirty to forty minutes. A small anterior proximal cavity should require not over one hour.

It would be better to make the larger proximal cavities, including those on the distal surface of cuspids, in two sittings. At the first sitting, the cavity is prepared but the cavosurface bevel is not finished; and, at the second sitting, the cavity is completed and filled. The first sitting should require one-half hour; the second sitting, one hour. Any filling requiring over one and one-half hours for completion should be filled by some other method. Foil fillings may be finished in most cases at the same sitting, thus saving both the patient's and the operator's time.

The fourth question usually asked by the patient is "How permanent is this filling going to be?"

The permanency of a filling depends more on the operator than the filling material used. The operator should study the occlusion and observe the location and condition of the soft tissues sur-

rounding the teeth. In cavity preparation, he should use judgment in locating the walls of the cavity so as to give the necessary strength to withstand the stress of mastication. Extension for prevention certainly should be the rule. All caries should be thoroughly removed from the cavity and the margins planed until no loosened enamel rods remain.

In the selection of a filling material, there are certain requirements which should be considered. The material should be capable of being thoroughly adapted and of remaining adapted to the walls of the cavity, effecting a seal of the margins. The material should not change in the fluids of the mouth and should be capable of retaining its contour and withstanding the stress of mastication. The material should not be irritating to the soft tissues and its margins should be finished flush with the tooth surface and remain intact over a long period of time. Gold foil, where indicated in present-day practice, comes nearer to meeting our needs, and we can promise our patients a filling that will be as nearly permanent as the tooth itself.

Because of the necessity for making contour restorations in treating smooth surface caries in the anterior teeth, cohesive gold foil is the type of foil which lends itself best to our requirements.

Our cavities should extend into the dentin and be prepared with sharp line and point angles and, wherever possible, with two parallel walls, such as in a gingival third cavity where the incisal and gingival walls may be paralleled.

In considering the cavity preparation on the proximal surfaces here, we have a triangular surface to consider, and therefore our preparation would have converging walls and would call for convenience points in the gingival for starting the filling and an incisal retention groove to prevent dislodgment.

In the manipulation of cohesive foil three principles are employed: welding, wedging and tempering.

It is possible to weld cold one piece of clean gold foil upon another. This may be done simply by burnishing. But in order to wedge the material thoroughly against the walls, the direction of force is an important consideration as is also the amount of force necessary to weld the foil thoroughly and counteract the elasticity of the dentin and pericemental membrane.

It has been clearly demonstrated that a pressure of 15 pounds on a plugger 1 mm. in diameter will condense gold foil. A smaller plugger will require a proportionately smaller amount of pressure.

This may be done by hand pressure, but it becomes a very tiresome process for the operator. Hand pressure is indicated in certain difficult points of access or where, for esthetic reasons, weakened walls have been permitted to remain.

In welding foil, the plugger should be moved in successive lines so that, with each step of the plugger, some uncondensed and some condensed filling is beneath the plugger at the same time. The direction of force should be changed according to the surface approached, driving the foil well into the point angles, line angles and against the dentin and enamel walls.

In order to temper foil, it is necessary to use successive blows of a mallet. The hand mallet used in the left hand by a competent assistant is a great aid to us. By this method, the operator is permitted to exert pressure with the plugger instruments before each blow is struck, thus minimizing the shock to the pericemental

membrane. Also, the time saved by having the assistant anneal and pass the foil is an important item.

Tempered gold foil has a tendency to spring, and this must be overcome in the manipulation of the material. In narrow cavities with two parallel walls, the gold is so thoroughly wedged into the elastic dentin walls that this tendency to spring is overcome.

When starting a filling in a point angle, this spring may be overcome by building the mass of foil in the form of a pyramid, following the three-legged-stool principle and then making any extensions in the form of a triangular bar, always building up a mass of gold to avoid the spring, which would be particularly noticeable in a thin sheet of tempered foil.

After-condensation, where indicated, usually consists in so tempering the filling that it will resist wear, be it the contact point or some surface coming under the stress of mastication. Repeated malleting with a small plugger point will give a very hard surface. This applies mainly to the lingual surface in the upper incisors where the angle is not involved. Where the angle is involved, the incisal portion of the filling should be well tempered as well as the lingual.

In the lower teeth, as the stress is on the labial surface, the labial portion of the filling is most thoroughly tempered. If an incisal angle is also restored, that portion too calls for after-condensation.

The finishing process consists in carving a foil restoration to contour with knives and files and, in some cases, using inverted cone stones. The margins of the filling and tooth surface should be made continuous. This is accomplished by the use of disks and strips. If a contact point is present, it should be properly located and then finished so that it will restore a ball contact with the adjacent tooth.

A dull finish may be accomplished by a slowly revolving rubber cup carrying moistened pumice to the surfaces requiring such treatment.

Just where should gold foil be then used in the anterior teeth in a present-day practice? Only in teeth where the roots are fully formed and no marked peridental disease is present; in lingual pits in lateral incisors; in small gingival third cavities, especially the upper and lower cuspids; in small anterior proximal cavities not involving the angle; in larger proximal cavities on the distal surfaces of the incisors and cuspids, and in restorations where there has been incisal wear and the incisal step would not be conspicuous.

A QUANTITATIVE AND QUALITATIVE CONSIDERATION OF THE BLOOD CELLS IN CASES WITH DENTAL INFECTION*

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WITH the presentation of additional evidence based upon careful scientific observation by such men as Hatton, Blaney, Hall, Rickert, Grove and others, on the successful treatment of teeth; and of Rosenow, Meisser, Price, Nichols, Haden and others on the incidence of systemic disturbances as the result of dental pathosis, including root canal treatment (good and bad alike), it becomes more of a perplexing problem to determine in a given instance when a particular tooth or group of teeth cease to be "safe" from the standpoint of the general health of the patient and become admitted liabilities. This has led some men to reason that extraction of such teeth should be done as a preventive measure, and others that it should be done only as a curative measure. The dentist must be constantly on the watch for diseased areas which might either jeopardize adjacent structures by local extension or act as foci by disseminating harmful bacteria and their byproducts to other parts of the body. Pockets around partially erupted teeth, those under restorative appliances and those formed as the result of pyorrhea or the presence of root fragments, unhealed areas following tooth removal, treated teeth or chronically infected and degenerated pulps may harbor infection, and these must be sought out and eradicated.

Each of the diagnostic procedures employed by the dentist in the search for such information has limits of usefulness. Roentgenography, the most universally used, brings to light only alterations in structure. Roentgenograms do not prove the presence of infection. Vitality tests merely arouse sensory response to electrical and thermal shock and frequently fail to afford the desired information. Percussion denotes tactile sensibility and intonation. Transillumination occasionally reveals opacity and congestion. Palpation may locate areas of fluctuation, tenderness or radiating pain, and probing only measure out accessible pockets and fistulae.

The conservative dentist is familiar with them all and uses each to correlate his visual inspection. There is a general tendency among diagnosticians to advise

*Read before the Section on Histology, Physiology, Pathology, Bacteriology and Chemistry (Research) at the Seventieth Annual Session of the American Dental Association, Minneapolis, Minn., Aug. 22, 1928.

*From the Hematological Laboratory, Department of Zoology, and Dental Division, Medical School, University of Minnesota. Hematological studies are under the direction and supervision of Hal Downey of the Department of Zoology. This work is being aided by a grant from the Scientific Foundation and Research Commission of the American Dental Association. The author is indebted to the officials of the university health service for access to records and assistance in obtaining material.

TABLE 1.—SUMMARY OF DENTAL FINDINGS[1]

	G	SP	MP	EP	PT	RRA	NA	F	CA	BR	PE	I	SO	NV
1					1		1							
12					4	1	3							
13					2		2							
16						1			1					
20						2			1					
22	X	X												
23	X		X											
24	X	X			3	1	2							1
26											4	1		
27					3	3					2	2		
30		X			1	X								
31					4	2	2							
32					3	3						4		
38					2		2							
40					1		1							
41					5	4	2				1			
47					1	1						4		
52												4		
56					3	2	1							1
61	X	X									4			
72					1		1	1						
73					2	2					2			
76	X				4	2	1							
80						1			1					
84					3		3				2			

1. In this table G indicates gingivitis; SP, slight pyorrhea; MP, moderate pyorrhea; EP, extreme pyorrhea; PT, treated teeth; RRA, radiolucent areas in roentgenogram; NA, no areas; F, fistulae; CA, cystic areas; BR, buried roots; SO, socket infection; PE, partially erupted teeth; I, impacted teeth; NV, negative to vitality tests.

removal of those treated teeth seen in roentgenograms which have radiolucent or radiopaque areas about them. These are regarded as probable menaces to general health, while those without such areas are not looked on with so much suspicion, except in association with systemic disease. Bacteriologic findings show that roentgenographically negative teeth give positive cultures and roentgenographically positive teeth give negative cultures in a large number of instances. This also adds to the general confusion.

TABLE 2.—HEMOGLOBIN

Class	1	2
Highest	97	94
Average	87 (plus)	82 (plus)
Lowest	78	72

TABLE 3.—RED BLOOD COUNT

Class	1	2
Highest	5,680,000	5,600,000
Average	4,951,742	4,666,935
Lowest	4,100,000	4,010,000

Laboratory workers in various fields have sought to utilize findings for the purpose of detecting the presence of mild chronic infection. Some have felt that their findings were of aid in the localizing of such areas.

DeNiord and Bixby[1] concluded that the uric acid content of the blood furnishes a reliable index to the complete elimination of foci of infection. On reexamination and against normal controls, Mueller[2] found the normal to vary through "considerable range" and that "retention is a positive factor only when a primary lesion in the kidneys can be demonstrated to exist." Price[3] has done considerable work on calcium utilization and disturbances for use in the treatment and prevention of dental disease. Its value is now not generally well understood. Zabriskie[4] reported a series of compliment fixation tests on dental cases.

TABLE 4.—TOTAL LEUKOCYTE COUNTS

Class	1	2
Highest	9,850	11,300
Average	8,427	8,406
Lowest	6,950	6,600

TABLE 5.—DISTRIBUTION OF LEUKOCYTE COUNTS

Counts	Class 1	Class 2
10—11,000	0	11%
9—10,000	18%	13%
8—9,000	66%	37%
7—8,000	11%	28%
6—7,000	5%	11%

These results have not been significant enough to be checked by others. Consid-

1. DeNiord, N. R., and Bixby, J. B.: Studies in Focal Infection, J. Lab. & Clin. Med., 7:573 (July) 1922.

2. Mueller, Emil: Blood Uric Acid in Relation to Focal Infection, J. A. D. A., 13:1232 (Sept.) 1926.

3. Price, W. A.: Newer Knowledge of Calcium Metabolism in Health and Disease, with Special Consideration of Calcification and Decalcification Processes, Including Focal Infection Phenomena, J. A. D. A., 13:1765 (Dec.) 1926.

4. Zabriskie, A.: Compliment Fixation Tests as Applied to Dental Practice, Dent. Cosmos, 62:201 (Feb.) 1920.

TABLE 6.—FINDINGS IN THIS SERIES

Case	Class	Red Blood Count	White Blood Count	Hemoglobin	Case	Class	Red Blood Count	White Blood Count	Hemoglobin
1	2	5,600,000-5,400,000	10,900-10,700	79-78	43	1	5,620,000-5,380,000	8,650- 8,450	89-88
2	1	5,320,000-5,130,000	9,650- 9,800	82-87	44	1	4,900,000-4,780,000	8,450- 8,600	87-91
3	1	4,400,000-4,380,000	8,550- 8,050	78-79	45	1	5,180,000-5,310,000	8,200- 8,550	93-95
4	1	5,360,000-5,360,000	9,400- 9,400	90-92	46	1	4,980,000-5,239,000	8,900- 9,000	90-86
5	1	6,010,000-4,870,000	8,500- 9,050	92-88	47	2	4,990,000-4,780,000	8,000- 7,890	89-91
6	1	5,180,000-5,290,000	9,550- 8,600	82-84	48	1	5,530,000-5,370,000	7,900- 8,250	86-89
7	1	4,880,000-4,860,000	6,900- 7,050	80-78	49	1	5,450,000-5,210,000	8,600- 8,050	87-90
8	1	5,130,000-5,030,000	8,550- 8,700	91-90	50	1	4,880,000-4,680,000	7,400- 7,090	78-80
9	1	4,720,000-4,750,000	8,850- 8,700	80-83	51	1	4,380,000-4,420,000	8,550- 8,350	82-84
10	1	5,100,000-4,980,000	9,750- 9,600	89-89	52	2	5,440,000-5,280,000	8,650- 8,800	87-88
11	1	5,030,000-5,050,000	8,800- 8,250	94-92	53	1	4,700,000-4,900,000	8,650- 8,250	87-90
12	2	4,250,000-4,340,000	9,100- 9,150	81-79	54	1	5,090,000-5,310,000	7,850- 7,090	85-88
13	2	4,890,000-4,780,000	9,050- 8,700	89-87	55	1	4,800,000-5,000,000	8,250- 7,900	84-87
14	1	4,290,000-4,100,000	7,300- 7,100	89-87	56	2	5,120,000-4,980,000	8,050- 8,400	80-80
15	1	5,200,000-5,210,000	8,000- 8,150	93-93	57	1	5,350,000-5,100,000	8,450- 8,800	88-91
16	2	4,490,000-4,560,000	6,650- 6,600	88-93	58	1	4,750,000-4,880,000	7,300- 7,000	79-83
17	1	4,690,000-4,610,000	8,450- 8,350	89-91	59	1	5,260,000-5,180,000	9,250- 8,800	83-95
18	1	4,300,000-4,240,000	6,850- 6,900	96-97	60	1	4,680,000-5,020,000	8,750- 8,300	84-88
19	1	5,260,000-5,240,000	7,400- 7,900	82-86	61	2	5,500,000-5,300,000	11,300-10,900	82-82
20	2	5,380,000-5,120,000	7,500- 7,600	90-88	62	1	5,320,000-5,130,000	9,750- 9,850	81-84
21	1	5,250,000-4,890,000	8,150- 8,300	87-88	63	1	4,350,000-4,380,000	8,450- 8,050	79-79
22	2	4,670,000-4,750,000	6,900- 6,850	81-79	64	1	5,340,000-5,360,000	9,010- 9,300	91-92
23	2	5,230,000-5,310,000	8,750- 8,900	85-87	65	1	5,110,000-4,970,000	8,450- 8,950	92-95
24	2	5,110,000-5,230,000	7,650- 7,450	94-92	66	1	5,170,000-5,290,000	9,450- 8,800	85-86
25	1	5,240,000-5,030,000	8,900- 8,750	88-89	67	1	4,970,000-4,720,000	6,950- 7,100	78-79
26	1	4,860,000-4,750,000	7,750- 7,500	89-91	68	1	5,010,000-5,230,000	8,400- 8,650	92-93
27	2	5,010,000-5,260,000	8,600- 8,400	94-93	69	1	4,890,000-5,370,000	9,600- 9,850	89-86
28	1	4,640,000-4,800,000	8,700- 8,700	92-90	70	1	4,710,000-4,860,000	8,900- 8,650	80-84
29	1	5,030,000-5,210,000	8,300- 8,650	87-84	71	1	5,470,000-5,680,000	8,750- 8,400	95-96
30	2	5,160,000-5,040,000	8,500- 8,250	86-88	72	2	4,400,000-4,180,000	10,050- 9,700	81-81
31	2	4,560,000-4,420,000	7,450- 7,200	79-80	73	2	4,750,000-4,960,000	8,950- 8,800	83-86
32	2	5,340,000-5,240,000	8,700- 8,900	91-93	74	1	4,480,000-4,220,000	7,250- 7,050	92-91
33	1	4,600,000-4,860,000	7,350- 7,500	88-84	75	2	4,680,000-4,490,000	7,000- 6,700	79-81
34	2	4,810,000-4,870,000	9,300- 8,750	92-94	76	1	5,380,000-5,090,000	8,000- 8,350	94-95
35	1	4,760,000-4,650,000	9,850- 9,500	89-89	77	1	4,590,000-4,760,000	8,500- 8,150	89-92
36	1	5,350,000-5,080,000	7,900- 7,750	91-86	78	1	4,260,000-4,480,000	6,750- 6,950	97-94
37	1	5,120,000-4,760,000	8,350- 8,100	90-93	79	1	5,090,000-5,370,000	8,750- 8,300	81-85
38	2	4,880,000-5,100,000	8,400- 8,250	84-87	80	2	5,260,000-5,190,000	7,500- 7,850	91-87
39	1	4,290,000-4,430,000	8,250- 8,600	85-81	81	1	6,070,000-4,810,000	8,050- 8,400	88-86
40	2	4,800,000-4,910,000	8,150- 8,450	83-84	82	2	4,730,000-4,010,000	7,300- 7,050	86-83
41	2	4,010,000-4,370,000	9,250- 8,850	72-74	83	2	5,480,000-5,070,000	7,200- 7,750	91-91
42	1	5,280,000-5,000,000	8,100- 8,400	90-87	84	1	5,000,000-5,430,000	8,950- 8,800	93-91
					85	2	4,560,000-4,370,000	8,350- 8,600	79-80

erable work has been done by those associated with institutions on the influence of dental infection on the number and proportion of the blood cells. Various groupings of material and lack of understanding of hematologic principles have led to findings which fail to agree.

I am attempting to check the validity of current claims regarding the practical value of hematologic findings to the dentist and to establish their value. The claim that there is a particular type of leukocyte formed in dental cases has now been thoroughly disproved. Further investigation consists of assigning the problem to its place in the fields of acute and chronic infection and determination of the importance it deserves.

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS

	No. 1	Class 1	No. 2	Class 1	No. 3	Class 1	No. 4	Class 1	No. 5	Class 1
NEUTROPHILS	No. 569	% 57.6	No. 600	% 57.4	No. 683	% 59.2	No. 662	% 61.5	No. 561	% 52.5
Myelocyte										
Toxic										
Band	31	5.4	7	0.2	2	2.9	15	2.3	1	0.2
Toxic band										
Segmented	497	87.2	506	85.8	654	95.8	628	94.9	543	98.6
Toxic seg.	12	2.2								
Fragile	29	5.2	87	14.0	27	3.9	19	2.9	17	3.0
LYMPHOCYTES	324	32.8	337	32.1	355	30.9	339	31.5	416	37.9
Micro	126	38.8	79	23.4	78	21.5	78	20.6	30	7.4
L. micro	3	0.9							1	0.2
Meso	88	27.1	192	56.9	167	47.0	180	53.1	277	66.7
L. meso	85	26.3	51	15.1	89	25.0	48	14.4	98	21.1
Macro	1	0.3	1	0.3	3	0.8	1	0.3	3	0.6
L. macro	4	1.2			2	0.5			1	0.2
Unclassified	17	5.2	14	4.1	17	4.8	32	9.4	6	1.2
MONOCYTES	66	6.6	80	7.6	67	5.6	48	4.5	70	6.8
Young	1	1.5	2	2.5	1	1.5				
Older			31	38.7	19	28.3	10	20.9	7	10.0
Toxic older	3	4.5								
Transitional	51	76.8	43	53.7	36	53.7	34	70.8	57	81.4
Toxic trans.	6	9.0								
Lobulated	3	4.5	4	5.0	11	16.4	4	8.3	6	8.5
Toxic lob.	2	3.0								
EOSINOPHILS	27	2.8	24	2.3	32	2.9	20	1.8	18	1.9
BASOPHILS	2	0.2	6	0.6	9	0.8	9	0.9	9	0.9
Total Cells Counted	988		1,047		1,147		1,074		1,074	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 6	Class 1	No. 7	Class 1	No. 8	Class 1	No. 9	Class 1	No. 10	Class 1
NEUTROPHILS	No. 654	% 60.1	No. 619	% 61.1	No. 670	% 62.4	No. 547	% 61.7	No. 652	% 63.0
Myelocyte					1	.18				
Toxic										
Band	21	3.2	9	1.4	19	2.8	17	3.2	24	3.6
Toxic band										
Segmented	618	92.9	594	95.9	632	94.3	511	93.1	604	92.6
Toxic seg.					4	0.6	11	2.0		
Fragile	15	2.3	16	2.6	14	2.1	8	1.4	24	3.6
LYMPHOCYTES	307	28.2	256	25.2	237	22.1	256	28.8	220	21.5
Micro	47	12.0	27	10.6	63	26.5	12	4.5	40	18.1
L. micro			1	0.4						
Meso	197	64.1	133	52.4	171	72.1	186	72.6	156	70.9
L. meso	62	20.2	70	27.3	51	21.5	42	12.5	48	21.8
Macro			9	3.5	1	0.4	7	2.3	4	1.8
L. macro	1	0.3	18	7.1	2	0.8	5	1.2		
Unclassified			25	10.2	12	5.0	16	6.2	12	5.5
MONOCYTES	95	8.7	76	7.5	69	6.4	58	6.5	92	9.0
Young	3	3.2	3	3.9	1	1.4			4	4.3
Older	26	27.4	12	15.6	14	20.2	7	12.1	12	11.8
Toxic older	54	56.7								
Transitional			54	73.7	38	55.0	42	72.4	32	35.8
Toxic trans.	12	12.5			2	2.8				
Lobulated			7	9.2	14	20.2	8	13.9	44	47.8
Toxic lob.							1	1.7		
EOSINOPHILS	25	2.3	27	2.6	21	1.9	11	1.2	4	0.4
BASOPHILS	7	0.7	9	0.9	12	1.1	2	0.2	4	0.4
Total Cells Counted	1,088		1,013		1,072		886		1,020	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 11	Class 1	No. 12	Class 2	No. 13	Class 2	No. 14	Class 1	No. 15	Class 1
NEUTROPHILS	No. 717	% 6.24	No. 580	% 59.0	No. 585	% 48.2	No. 587	% 53.5	No. 697	% 63.2
Myelocyte									1	0.14
Toxic										
Band	31	4.3	41	7.0	19	3.2	12	2.0	8	1.2
Toxic band					1	0.1				
Segmented	656	91.9	497	85.6	518	88.5	555	94.5	661	94.8
Toxic seg.					7	1.1				
Fragile	27	3.7	32	5.5	40	6.8	30	5.1	27	3.9
LYMPHOCYTES	354	30.8	268	27.2	297	29.5	270	24.6	288	26.1
Micro	51	14.4	59	22.0	89	29.9	48	17.8	61	21.2
L. micro									3	1.0
Meso	236	66.6	72	26.8	96	32.3	186	68.8	172	59.7
L. meso	26	7.3	111	41.4	92	30.9	60	22.2	29	10.1
Macro			2	0.74					3	1.0
L. macro			7	2.6	2	0.6			2	0.7
Unclassified	41	11.6	17	6.3	18	6.0	36	13.3	19	6.6
MONOCYTES	57	4.9	86	8.7	75	7.4	70	6.3	69	6.6
Young			3	3.4					1	1.4
Older	6	10.5	41	47.6	33	44.0	16	22.8	32	46.3
Toxic older					4	5.3			2	2.8
Transitional	37	64.9	21	24.4	29	38.6	27	38.5	14	20.2
Toxic trans.			8	9.3	2	2.6				
Lobulated	14	24.5	11	12.7	7	9.3	27	38.5	19	27.5
Toxic lob.			2	2.3	0				1	1.4
EOSINOPHILS	14	1.2	19	1.9	37	3.6	5	0.45	31	2.8
BASOPHILS	6	0.5	30	3.0	12	1.1	4	0.36	17	1.5
Total Cells Counted	1,148		983		1,006		1,096		1,102	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 16	Class 2	No. 17	Class 1	No. 18	Class 1	No. 19	Class 1	No. 20	Class 2
NEUTROPHILS	No. 563	% 56.8	No. 518	% 53.3	No. 654	% 60.5	No. 466	% 48.1	No. 515	% 53.2
Myelocyte										
Toxic										
Band	63	11.0	14	2.7	6	0.91	35	7.5	21	4.0
Toxic band	8	1.1							11	1.9
Segmented	404	73.6	406	78.4	612	93.6	411	88.1	386	75.0
Toxic seg.	17	3.0					6	1.2	59	11.4
Fragile	71	12.6	98	18.9	36	5.5	14	2.7	38	7.4
LYMPHOCYTES	301	30.4	378	38.9	310	22.8	367	33.9	348	35.9
Micro	93	30.9	56	14.8	66	21.3	123	33.5	119	3.4
L. micro							4	1.1		
Meso	53	17.6	147	38.9	240	77.4	180	49.0	74	34.2
L. meso	126	42.8	105	27.7	18	5.8	11	3.0	86	24.7
Macro	2	0.7	3	0.7						
L. macro	5	1.6	6	1.5			4	1.1	7	2.2
Unclassified	22	7.3	63	16.6	6	1.9	45	12.3	62	17.9
MONOCYTES	87	8.7	56	5.7	61	5.6	64	5.9	70	7.2
Young	2	2.2								
Older	12	13.7	7	12.5			2	3.1	13	18.6
Toxic older	3	3.4							5	7.2
Transitional	34	39.0	35	62.5	30	49.2	19	29.7	41	58.6
Toxic trans.	7	8.0							3	4.8
Lobulated	26	29.8	14	25.0	31	50.8	43	61.2	6	8.5
Toxic lob.	3	3.4							2	2.9
EOSINOPHILS	28	2.8	21	2.1	23	2.1	16	1.5	24	2.5
BASOPHILS	13	1.3	0	0	12	0.11	4	0.4	11	1.2
Total Cells Counted	992		973		1,080		1,081		968	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 21	Class 1	No. 22	Class 2	No. 23	Class 2	No. 24	Class 2	No. 25	Class 1
NEUTROPHILS	No. 574	% 54.4	No. 598	% 58.3	No. 602	% 59.2	No. 683	% 61.0	No. 640	% 61.0
Myelocyte										
Toxic										
Band	36	6.2	15	2.5	17	2.8	7	1.1	10	1.5
Toxic band			7	1.1	6	1.0	2	0.3		
Segmented	484	84.3	521	87.0	468	77.7	586	85.8	600	93.8
Toxic seg.			37	6.2	30	4.9	21	3.1		
Fragile	54	9.4	18	26.9	81	13.5	67	9.8	30	4.6
LYMPHOCYTES	323	30.6	277	26.9	328	32.3	304	27.1	331	31.5
Micro	41	12.7	81	29.2	181	55.2	86	28.3	55	16.6
L. micro					3	0.9				
Meso	183	56.7	98	35.3	56	17.2	121	39.8	181	54.6
L. meso	51	15.8	68	24.1	42	12.8	34	11.2	17	5.1
Macro			1	0.4	1	0.3				
L. macro			2	0.7	3	0.9	2	0.6		
Unclassified	48	14.8	27	9.7	42	12.8	61	2.1	78	23.6
MONOCYTES	107	10.1	112	10.9	61	6.0	89	7.1	61	78
Young							3	3.3		
Older	27	25.2	19	16.9	6	9.8	24	27.0	5	19.2
Toxic older			3	0.3	2	3.3				
Transitional	23	21.5	81	72.2	43	70.5	52	58.4	30	35.9
Toxic trans.			7	6.3	3	4.9	3	3.4		
Lobulated	57	53.3	2	1.7	7	11.5	7	7.9	16	20.5
Toxic lob.										
EOSINOPHILS	49	4.9	32	3.1	7	0.7	31	3.2	15	1.4
BASOPHILS	2	0.1	9	0.9	8	0.8	12	1.3	4	0.38
Total Cells Counted	1,055		1,028		1,016		1,119		1,050	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 26	Class 2	No. 27	Class 2	No. 28	Class 1	No. 29	Class 1	No. 30	Class 2
NEUTROPHILS	No. 647	% 56.5	No. 668	% 50.8	No. 707	% 71.7	No. 481	% 47.9	No. 62.2	% 58.4
Myelocyte									1	0.2
Toxic										
Band	18	2.6	17	2.6	30	4.2	4	0.83	21	3.4
Toxic band	3	0.4								
Segmented	583	90.4	569	85.2	657	92.9	462	96.1	509	81.8
Toxic seg.	11	1.7	37	5.5					31	4.9
Fragile	32	4.9	42	6.3	20	2.8	14	2.9	60	9.7
LYMPHOCYTES	386	33.8	303	27.1	221	22.4	409	40.8	314	28.6
Micro	72	18.7	78	25.7	51	23.0	83	20.3	96	30.6
L. micro									1	0.3
Meso	203	52.6	69	22.4	52	23.5	226	55.3	107	34.1
L. meso	74	19.2	87	28.7	90	40.7	53	12.9	69	22.0
Macro			1	0.3						
L. macro			2	0.6	3	1.3			2	0.6
Unclassified	37	9.58	66	21.7	25	11.3	47	11.5	40	12.7
MONOCYTES	66	5.7	107	9.5	41	4.5	110	10.9	101	9.5
Young	2	3.0	4	3.7			6	5.45		
Older	10	15.2	19	17.7	20	61.1	21	19.1	16	15.8
Toxic older			2	1.8					1	0.9
Transitional	28	42.4	69	64.5	11	26.8	46	41.8	61	60.4
Toxic trans.			8	7.5					4	3.9
Lobulated	26	39.4	4	3.6	10	24.4	37	33.6	17	16.8
Toxic lob.			1	0.9			2	0.2		
EOSINOPHILS	7	0.61	28	2.5	10	1.0	16	1.6	22	2.0
BASOPHILS	5	0.43	12	1.0	2	0.2	12	1.1	6	0.6
Total Cells Counted	1,144		1,118		986		1,027		1,065	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 31	Class 2	No. 32	Class 2	No. 33	Class 1	No. 34	Class 1	No. 35	Class 1
NEUTROPHILS	No. 560	% 55.8	No. 645	% 62.	No. 531	% 51.8	No. 760	% 68.0	No. 594	% 64.8
Myelocyte										
Toxic										
Band	17	3.0	12	1.8	17	3.2	47	6.19	11	1.85
Toxic band										
Segmented	489	87.3	583	90.3	495	97.6	652	85.8	560	94.4
Toxic seg.	7	1.2	12	1.8						
Fragile	47	8.4	38	5.9	19	3.5	61	7.9	23	3.8
LYMPHOCYTES	342	34.1	300	29.0	371	36.3	269	24.0	258	28.1
Micro	103	30.1	92	30.3	101	27.2	34	12.6	74	28.7
L. micro	1	0.3								
Meso	69	20.2	107	35.0	193	52.0	142	52.8	132	51.2
L. meso	154	45.0	69	23.7	46	12.4	57	21.7	44	17.0
Macro	2	0.6			3	0.8				
L. macro	1	0.3	3	1.2						
Unclassified	12	3.5	29	9.0	28	7.5	36	13.4	8	31.0
MONOCYTES	70	6.9	70	6.9	79	7.71	67	6.0	47	51.2
Young			2	2.9						
Older	7	10.0	17	24.3			11	16.4	5	10.6
Toxic older										
Transitional	49	70.0	38	54.3	56	70.9	15	22.4	13	27.6
Toxic trans.	3	4.2	9	12.9						
Lobulated	11	15.7	4	5.7	23	29.1	41	61.2	29	61.7
Toxic lob.										
EOSINOPHILS	31	3.1	14	1.4	32	3.12	19	1.7	17	1.82
BASOPHILS	8	0.8	6	0.6	11	1.0	2	0.1	1	0.1
Total Cells Counted	1,011		1,030		1,024		1,117		917	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 36	Class 1	No. 37	Class 1	No. 38	Class 2	No. 39	Class 1	No. 40	Class 2
NEUTROPHILS	No. 616	% 57.8	No. 625	% 54.7	No. 658	% 58.4	No. 562	% 57.2	No. 596	% 57.7
Myelocyte					1	0.2				
Toxic									1	0.2
Band	7	1.0	1	0.16	11	1.7	14	2.4	8	1.4
Toxic band					1	0.2	2	0.3	1	0.2
Segmented	592	96.1	613	98.1	592	89.9	518	92.2	537	92.0
Toxic seg.					37	5.6	7	1.2	12	2.2
Fragile	17	2.8	11	1.7	16	2.4	21	3.7	37	4.6
LYMPHOCYTES	369	34.6	365	81.9	361	32.1	360	36.6	329	31.8
Micro	120	32.5	160	43.8	68	18.8	142	39.4	83	25.2
L. micro							1	0.27		
Meso	216	5.9	141	38.6	113	31.0	108	30.0	69	20.9
L. meso	2	54.2	22	6.0	147	40.8	76	21.0	153	46.5
Macro							3	0.8		
L. macro					12	3.4	3	0.8	8	2.4
Unclassified	31	8.4	42	11.5	21	6.0	27	7.5	16	4.8
MONOCYTES	49	4.6	127	11.1	80	7.1	50	5.0	56	5.4
Young			6	4.7			2	0.5	1	1.8
Older			32	25.2	34	42.5	8	16.0	12	21.5
Toxic older					9	10.2				
Transitional	31	63.3	26	20.5	26	32.5	23	46.0	37	66.1
Toxic trans.					3	3.8	2	5.0	4	7.1
Lobulated	18	36.7	31	24.4	8	10.0	14	28.0	2	3.6
Toxic lob.							1	2.0		
EOSINOPHILS	29	2.7	14	1.21	28	2.5	9	0.9	37	3.6
BASOPHILS	3	0.3	11	0.9	4	0.3	3	0.2	14	1.3
Total Cells Counted	1,066		1,143		1,127		983		1,032	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 41	Class 2	No. 42	Class 1	No. 43	Class 1	No. 44	Class 1	No. 45	Class 1
NEUTROPHILS	No. 688	% 70.6	No. 705	% 65.2	No. 692	% 68.5	No. 656	% 60.6	No. 656	% 59.4
Myelocyte					2	0.2	1	1.5		
Toxic										
Band	16	2.3	3	0.4	21	3.0	33	5.03	8	1.2
Toxic band										
Segmented	648	94.1	684	97.0	631	91.2	582	88.7	621	94.7
Toxic seg.	12	1.7	1	0.1	7	1.0	2	0.3	3	0.47
Fragile	13	1.9	17	2.4	31	4.4	38	5.7	24	3.6
LYMPHOCYTES	253	25.9	278	25.7	227	22.3	321	29.6	335	30.3
Micro	102	40.4	47	16.9	56	24.7	102	31.8	86	25.7
L. micro	3	1.2			2	0.8	3	0.9		
Meso	69	27.3	136	48.9	122	42.0	162	50.5	184	54.9
L. meso	42	16.6	78	28.1	28	12.3	19	5.9	37	11.0
Macro			1	0.3	7	3.0	3	0.9		
L. macro	1	0.4	2	0.7	3	1.3	1	0.3	2	0.5
Unclassified	36	14.2	14	5.0	9	3.9	31	9.6	26	7.7
MONOCYTES	105	10.9	65	6.0	65	6.3	87	8.0	68	6.1
Young							3	3.3		
Older	19	18.1	21	32.3	14	21.5	9	10.5	12	17.6
Toxic older	1	0.9	1	1.5						
Transitional	68	64.8	31	47.7	46	70.8	68	78.2	46	67.7
Toxic trans.	3	2.9			2	3.4	2	2.2	1	1.6
Lobulated	14	14.3	12	18.4	3	4.5	4	4.6	9	13.2
Toxic lob.							1	1.1		
EOSINOPHILS	16	1.7	26	2.4	31	3.0	8	0.7	31	2.8
BASOPHILS	11	1.12	8	0.7	2	0.1	10	0.9	14	1.2
Total Cells Counted	974		1,082		1,017		1,082		1,104	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 46	Class 1	No. 47	Class 2	No. 48	Class 1	No. 49	Class 1	No. 50	Class 1
NEUTROPHILS	No. 575	% 60.5	No. 590	% 59.2	No. 601	% 60.6	No. 651	% 64.8	No. 669	% 65.4
Myelocyte	1	0.17							1	0.2
Toxic										
Band	4	0.6	41	7.1	14	2.3	7	1.0	9	1.3
Toxic band			3	0.5			2	0.3		
Segmented	491	85.4	448	76.0	567	94.3	623	95.7	621	92.8
Toxic seg.	17	2.9	52	8.7	2	0.3	1	0.1	7	1.0
Fragile	62	10.8	46	7.2	18	3.0	28	4.3	31	4.5
LYMPHOCYTES	295	30.3	304	30.6	317	32.0	301	30.0	293	29.3
Micro	108	36.6	109	35.9	63	19.9	47	15.3	56	19.1
L. micro	1	0.3					1	0.3	1	0.3
Meso	114	47.5	97	34.4	209	66.0	148	49.2	163	59.7
L. meso	26	8.8	73	24.7	29	9.1	82	27.3	42	14.4
Macro					4	1.2	1	0.3	1	0.3
L. macro	8	2.7	4	15.4	2	0.6	1	0.3	2	0.7
Unclassified	38	12.9	21	6.7	11	3.4	21	6.9	28	9.6
MONOCYTES	65	6.8	82	8.2	64	6.4	63	6.2	53	5.5
Young	1	1.5					2	3.1		
Older	18	27.7	20	24.4	9	14.1	19	30.2	2	3.7
Toxic older			2	2.4						
Transitional	32	49.3	47	57.3	46	71.9	32	50.8	36	68.0
Toxic trans.	3	4.6	8	9.8			1	1.5	1	1.8
Lobulated	11	16.9	4	4.8	2	3.1	8	12.7	14	26.5
Toxic lob.			1	1.2	6	9.3	1	1.5		
EOSINOPHILS	17	1.7	14	1.5	7	0.71	13	1.3	7	0.6
BASOPHILS			7	0.7	2	0.2			2	0.2
Total Cells Counted	952		997		991		1,028		1,024	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 51	Class 1	No. 52	Class 2	No. 53	Class 1	No. 54	Class 1	No. 55	Class 1
NEUTROPHILS	No. 599	% 61.1	No. 648	% 63.4	No. 580	% 57.4	No. 565	% 60.3	No. 622	% 60.3
Myelocyte										
Toxic										
Band	3	0.5	17	2.9	28	4.8	11	1.9	3	0.4
Toxic band					2	0.3				
Segmented	563	94.0	602	91.0	496	85.5	539	95.5	593	95.4
Toxic seg.	21	3.5	3	0.5	17	2.7	3	0.4		
Fragile	12	2.0	26	4.1	37	6.4	12	2.1	26	4.1
LYMPHOCYTES	278	28.4	279	2.63	321	31.7	289	30.8	306	29.7
Micro	123	44.2	130	48.6	82	25.5	29	10.1	54	17.7
L. micro	4	1.4								
Meso	99	35.6	74	22.9	76	2.3	114	39.4	166	54.3
L. meso	32	11.5	53	19.0	118	36.8	106	36.7	72	23.5
Macro			1	0.4	3	0.9	7	2.43		
L. macro	6	2.1	2	0.8	8	2.4	14	4.8	2	65.4
Unclassified	14	5.0	19	6.9	34	10.6	19	3.1	12	3.85
MONOCYTES	64	6.5	66	6.4	68	6.7	57	6.0	53	5.1
Young					2	2.9	1	1.7		
Older	13	16.2	–4	6.6	12	17.3	3	5.2	7	13.4
Toxic older	2	3.1							1	1.8
Transitional	40	62.5	52	78.8	51	73.3	42	73.7	32	60.4
Toxic trans.			3	4.5			2	3.5	1	1.8
Lobulated	9	14.1	7	10.5	2	2.9	9	15.8	12	22.6
Toxic lob.					1	1.4				
EOSINOPHILS	31	3.1	16	1.5	26	2.5	18	1.9	32	3.1
BASOPHILS	8	0.8	12	1.1	16	1.5	7	0.7	9	0.8
Total Cells Counted	980		1,021		1,011		936		1,022	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 56	Class 2	No. 57	Class 1	No. 58	Class 1	No. 59	Class 1	No. 60	Class 1
NEUTROPHILS	No. 549	% 58.9	No. 637	% 59.6	No. 623	% 60.8	No. 655	% 69.8	No. 670	% 62.8
Myelocyte									1	0.2
Toxic										
Band	14	3.3	22	3.4	7	1.1			18	2.7
Toxic band	2	3.6								
Segmented	429	78.3	601	94.4	563	90.4	572	87.4	633	94.5
Toxic seg.	68	7.7			12	1.9			5	0.7
Fragile	36	6.7	14	2.2	41	6.5	83	12.6	13	1.9
LYMPHOCYTES	371	36.0	330	30.9	288	28.1	227	24.2	296	27.7
Micro	82	22.1	27	8.1	30	10.4	41	18.1	62	24.6
L. micro							1	0.4		
Meso	141	38.1	143	34.4	208	72.2	123	54.2	170	57.4
L. meso	106	28.6	125	38.2	39	13.5	51	22.5	41	13.9
Macro	4	1.1	9	2.7					2	0.7
L. macro	9	2.6	12	3.6			2	0.8	7	2.4
Unclassified	29	7.8	13	3.9	11	3.9	9	3.9	14	4.7
MONOCYTES	83	8.1	73	6.8	65	6.3	46	4.9	64	6.0
Young	3	3.4	4	5.4					1	1.6
Older	18	21.6	11	15.1	15	23.1	24	52.2	12	18.2
Toxic older	1	1.2								
Transitional	52	62.6	37	51.0	41	63.1	21	45.6	36	56.2
Toxic trans.	3	3.6			2	3.1				
Lobulated	4	4.8	19	26.0	7	10.6	1	2.2	15	2.3
Toxic lob.	2	2.4	2	2.4						
EOSINOPHILS	9	0.9	14	1.3	36	3.5	8	0.8	30	2.8
BASOPHILS	12	1.1	4	0.3	12	1.1	2	0.2	7	0.6
Total Cells Counted	1,024		1,058		1,024		938		1,067	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 61	Class 2	No. 62	Class 1	No. 63	Class 1	No. 64	Class 1	No. 65	Class 1
NEUTROPHILS	No. 670	% 62.8	No. 533	% 57.1	No. 651	% 65.4	No. 708	% 69.2	No. 662	% 63.2
Myelocyte	1	0.2								
Toxic										
Band	18	2.7	17	3.1	23	3.5	29	4.1	8	1.2
Toxic band										
Segmented	633	94.5	496	93.1	604	92.8	658	92.9	624	94.2
Toxic seg.	5	0.7							9	1.3
Fragile	13	1.9	20	3.8	24	3.6	21	2.9	21	3.1
LYMPHOCYTES	296	27.7	310	33.2	248	24.9	232	22.7	297	28.3
Micro	62	24.6	103	33.2	30	12.1	15	6.4	57	19.1
L. micro									2	0.6
Meso	170	57.4	186	60.0	159	64.1	107	46.1	165	55.5
L. meso	41	13.9	13	4.9	39	15.7	86	37.1	39	13.1
Macro	2	0.7							2	0.6
L. macro	7	2.4			5	2.0	7	3.0	1	0.3
Unclassified	14	4.7	8	2.6	15	6.0	17	7.3	31	10.5
MONOCYTES	64	6.0	71	7.6	71	7.1	50	4.8	57	5.3
Young	1	1.6							1	1.7
Older	12	18.2	6	8.4	6	8.4	21	42.0	26	45.6
Toxic older							3	6.0		
Transitional	36	56.2	43	60.6	61	85.9	12	24.0	22	38.6
Toxic trans.			1	1.4	3	4.2	4	8.0	1	1.7
Lobulated	15	2.3	21	29.6	1	1.4	9	18.0	7	12.3
Toxic lob.							1	2.0		
EOSINOPHILS	30	2.8	19	2.0	14	1.4	29	2.8	18	1.7
BASOPHILS	7	0.6	1	0.1	11	1.1	4	0.3	14	1.3
Total Cells Counted	1,067		934		995		1,023		1,048	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 66	Class 1	No. 67	Class 1	No. 68	Class 1	No. 69	Class 1	No. 70	Class 1
NEUTROPHILS	No. 572	% 64.2	No. 539	% 56.7	No. 604	% 63.6	No. 696	% 65.0	No. 576	% 59.2
Myelocyte							1	0.15		
Toxic										
Band	36	6.2	13	2.4	9	1.5	4	0.5		
Toxic band										
Segmented	484	84.7	428	79.4	583	96.5	608	87.3	562	97.6
Toxic seg.							12	1.7		
Fragile	52	9.1	98	18.2	12	19.4	71	10.1	14	2.4
LYMPHOCYTES	235	26.4	347	36.5	257	27.1	308	28.7	301	31.0
Micro	39	16.6	55	15.7	21	8.2	41	13.5	123	40.9
L. micro										
Meso	114	48.5	105	30.2	170	66.5	183	59.4	87	28.9
L. meso	61	26.0	147	42.4	33	17.1	71	23.0	68	2.2
Macro	7	2.9			7	2.7				
L. macro	3	1.2			12	4.6	1	0.3	4	13.3
Unclassified	11	4.6	40	11.5	14	5.4	12	3.8	19	6.31
MONOCYTES	57	6.4	44	4.6	56	5.9	52	4.9	71	7.3
Young					1	1.7	2	3.8		
Older	24	42.1	6	13.6	12	21.4	21	40.3	9	12.7
Toxic older										
Transitional	27	47.4	32	72.7	42	73.0	20	38.5	48	67.6
Toxic trans.	3	5.2					2	3.9		
Lobulated	2	3.5	6	1.3	3	5.3	7	13.5	14	19.7
Toxic lob.	1	1.7								
EOSINOPHILS	20	2.2	12	1.2	24	2.5	13	1.2	14	1.4
BASOPHILS	7	0.7	8	0.8	9	0.9	2	0.2	10	1.0
Total Cells Counted	891		950		950		1,071		972	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 71	Class 1	No. 72	Class 2	No. 73	Class 2	No. 74	Class 1	No. 75	Class 1
NEUTROPHILS	No. 483	% 53.6	No. 573	% 57.1	No. 708	% 63.3	No. 580	% 56.4	No. 657	% 60.1
Myelocyte										
Toxic										
Band	4	0.8	26	4.5	19	2.9	6	1.1	3	0.6
Toxic band			2	0.3						
Segmented	460	95.3	499	87.1	662	93.5	536	92.2	603	91.7
Toxic seg.			14	2.4	8	1.4	2	0.3	5	1.0
Fragile	19	3.9	34	5.9	19	2.9	36	6.2	26	3.9
LYMPHOCYTES	348	38.6	339	32.7	311	27.8	354	34.4	338	30.9
Micro	81	23.2	122	33.1	67	21.5	93	26.2	89	26.3
L. micro			4	1.1						
Meso	202	59.8	86	25.3	189	60.7	131	36.7	96	28.4
L. meso	48	13.7	93	27.3	37	11.6	96	21.4	114	33.8
Macro	3	0.8	2	0.5					1	0.3
L. macro			8	2.3	2	0.6			4	1.2
Unclassified	14	4.3	24	7.3	16	5.1	59	16.6	34	10.1
MONOCYTES	54	6.0	88	8.7	75	6.7	54	5.2	62	5.7
Young										
Older	20	37.1	34	38.6	18	24.0	12	22.2	20	32.3
Toxic older			3	3.4						
Transitional	21	38.8	43	48.8	51	68.0	36	66.6	35	56.5
Toxic trans.	4	7.4	6	6.8	3	0.4	1	1.8		
Lobulated	9	16.6	2	2.2	2	2.6	5	9.2	7	11.3
Toxic lob.					1	1.3				
EOSINOPHILS	12	1.4	36	3.5	14	1.2	26	2.5	41	3.8
BASOPHILS	3	0.3	9	0.9	11	1.0	8	0.6	16	1.5
Total Cells Counted	900		1,004		1,119		1,027		1,094	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 76	Class 2	No. 77	Class 1	No. 78	Class 1	No. 79	Class 1	No. 80	Class 2
NEUTROPHILS	No. 644	% 59.5	No. 580	% 56.6	No. 653	% 59.6	No. 598	% 59.9	No. 557	% 59.3
Myelocyte										
Toxic							11	1.8	26	4.7
Band	20	3.1	11	1.9	4	0.6				
Toxic band							554	92.6	493	88.5
Segmented	537	83.4	536	92.5	597	91.1				
Toxic seg.	48	7.5	7	1.2						
Fragile	39	6.0	26	4.5	52	8.3	33	5.5	38	6.8
LYMPHOCYTES	322	29.7	384	36.5	348	31.8	321	3.22	273	29.1
Micro	68	21.1	128	33.3	45	12.9	52	16.4	59	24.5
L. micro	2	0.6								
Meso	107	33.2	77	20.1	186	53.4	173	53.8	72	26.3
L. meso	119	36.9	153	39.9	89	25.6	62	19.3	121	44.6
Macro	3	0.9							1	36.6
L. macro	9	2.8	4	1.1					6	2.5
Unclassified	14	4.3	22	5.9	28	6.3	34	10.5	14	5.0
MONOCYTES	84	7.8	56	5.6	70	6.4	59	5.9	72	7.7
Young	2	2.4							4	5.6
Older	19	22.6	12	21.5	9	12.9	14	23.7	32	4.4
Toxic older										
Transitional	53	62.1	34	60.7	47	67.1	26	44.1	20	7.8
Toxic trans.	7	8.3	2	3.6					4	5.5
Lobulated			8	14.2	12	17.2	19	32.2	9	12.5
Toxic lob.	3	4.7			2	2.9			3	4.1
EOSINOPHILS	22	2.0	19	1.8	10	0.9	15	1.6	23	2.4
BASOPHILS	8	0.7	12	1.1	14	1.3	5	0.6	14	1.5
Total Cells Counted	1,080		1,051		1,095		998		939	

TABLE 7.—QUALITATIVE (MORPHOLOGIC) ANALYSIS OF THE BLOOD CELLS—*Continued*

	No. 81	Class 1	No. 82	Class 2	No. 83	Class 1	No. 84	Class 1	No. 85	Class 1
NEUTROPHILS	No. 702	% 61.1	No. 590	% 59.4	No. 683	% 59.1	No. 576	% 57.5	No. 623	% 61.1
Myelocyte										
Toxic	32	4.5								
Band	2	0.4	18	3.1	12	1.8	7	1.2	10	1.6
Toxic band	647	92.1	2	0.4						
Segmented			521	88.3	642	93.1	543	94.1	587	94.2
Toxic seg.			6	1.2						
Fragile	23	3.2	43	7.3	27	3.9	26	4.5	26	4.7
LYMPHOCYTES	340	29.6	303	30.5	368	31.9	340	33.9	287	28.2
Micro	72	21.2	87	28.7	48	13.1	130	38.2	89	31.0
L. micro										
Meso	183	53.9	94	31.1	219	56.9	39	11.8	126	43.9
L. meso	69	20.3	97	32.1	81	22.1	108	31.2	48	16.7
Macro	1	0.3			4	1.1			3	1.1
L. macro	3	0.9	6	1.9			2	0.6	2	0.7
Unclassified	12	3.6	19	6.3	16	4.4	61	17.4	19	6.4
MONOCYTES	77	6.7	79	7.9	54	4.7	53	5.2	84	8.2
Young										
Older	16	20.9	32	40.5	6	11.2	10	18.8	4	4.7
Toxic older			5	6.4						
Transitional	54	70.1	26	33.0	36	66.7	36	67.9	73	86.8
Toxic trans.			3	3.9	2	3.7	2	0.4		
Lobulated	7	9.1	9	11.4	9	16.7	5	9.2	6	7.2
Toxic lob.			4	5.1	1	1.8			1	1.2
EOSINOPHILS	19	1.7	14	1.4	32	2.8	21	2.0	18	1.8
BASOPHILS	12	1.0	8	0.9	19	1.7	13	1.2	7	0.7
Total Cells Counted	1,150		994		1,156		1,006		1,019	

TABLE 8.—COMPARATIVE SUMMARY OF THE QUALITATIVE DIFFERENTIAL COUNTS

	Class 1			Class 2		
	Average	Highest	Lowest	Average	Highest	Lowest
Neutrophil	**59.5**	**71.7**	**47.9**	**54.7**	**70.6**	**48.1**
Myelocyte	0.01	0.2	0	0.01	0.2	0
Toxic myelocyte	0	0	0	1 cell		
Band	2.3	6.9	0.0	3.9	9.0	1.1
	(In 95% of cases)			(In 96% of cases)		
Toxic band	0.1	0.3	0.0	0.4	1.9	0
	(In 5% of cases)			(In 52% of cases)		
Segmented	91.9	98.6	76.0	84.9	94.1	73.6
Toxic seg	0.3	3.0	0.0	3.4	11.4	0.0
	(In 84% of cases)			(In 92% of cases)		
Fragile	5.3	19.2	1.4	6.2	13.5	1.9
Lymphocytes	**32.1**	**40.8**	**21.5**	**34.6**	**36.0**	**25.9**
Micro	23.2	48.8	4.5	30.3	48.6	18.7
L. micro	0.1	1.4	0.0	0.2	1.2	0.0
	(In 23% of cases)			(In 24% of cases)		
Meso	50.9	77.4	11.8	32.3	60.7	17.2
L. meso	21.6	54.2	5.8	27.5	46.5	4.9
Macro	0.6	3.5	0.0	0.2	1.1	0.0
	(In 50% of cases)			(In 48% of cases)		
L. macro	1.0	7.1	0.0	1.4	3.4	0.3
	(In 63% of cases)			(In 96% of cases)		
Unclassed	8.4	31.0	1.9	8.1	21.7	2.1
Monocytes	**6.3**	**11.1**	**4.5**	**7.7**	**10.9**	**5.4**
Young	0.9	5.4	0.0	1.3	5.6	0.0
	(In 35% of cases)			(In 44% of cases)		
Older	24.0	61.1	3.1	26.1	47.6	6.6
Toxic older	0.2	6.0	0.0	1.8	10.2	0.0
	(In 8% of cases)			(In 51% of cases)		
Transitional	55.5	86.8	20.5	52.7	78.8	24.4
Toxic trans	4.4	8.0	0.4	6.0	12.9	1.4
	(In 35% of cases)			(In 96% of cases)		
Lobulated	18.3	61.7	1.3	11.1	39.4	1.7
Toxic lob	1.5	8.0	0.4	6.0	12.9	1.4
	(In 23% of cases)			(In 44% of cases)		
Eosinophil	**1.9**	**4.9**	**0.4**	**2.1**	**3.6**	**0.4**
Basophil	**0.7**	**1.7**	**0.0**	**0.9**	**3.0**	**0.1**

The object of this paper is to report a study of the hemoglobin values, red cell counts and white cell counts (Table 6) and a morphologic analysis of the blood cells (Table 7) found in a group of young men who were as free from all types of ailments as it is practically possible to determine, and to compare these with a similar group having dental disturbances of various types (Table 1). This was done to determine the effect of dental infection alone on the number, proportion and histology of the blood cells from a carefully selected group of normally healthy adults at an age when the physical power to oppose untoward influences is unquestionably at its height. The first of these groups will be referred to as Class 1 and the latter as Class 2.

The literature, selection of material and methods have been presented in previous papers.[5] As controllable factors

5. Epstein, I. A.: Study of the Blood in Cases with Dental Infection: Preliminary Report, J. A. D. A., 13:1752 (Dec.) 1926; Qualitative and Quantitative Consideration of the Cells in Normal Blood, ibid., 15:1055 (June) 1928.

accountable for variation in the blood picture have been considered also, they will not be reviewed here, except as referred to in the discussion. This report is based on sixty of Class 1 cases and twenty-five of Class 2.

HEMOGLOBIN AND RED CELLS

The hemoglobin values (Table 2) in Class 1 cases range from 78 to 97 per cent (Sahli), the numerical average being 87 per cent plus. The values for Class 2 cases extend from 72 to 94 per cent, having a numerical average of 82 per cent plus. It is of interest to note that although the average is five points lower, the hemoglobin readings for Class 2 cases, with the exception of Case 41, all lie between the limits of the Class 1, normal group.

The variation in the red cell counts (Table 3) also correspond to this change. Those in Class 1 range from 4,100,000 to 5,600,000, with an average of 4,951,742, and in Class 2 cases, from 4,010,000 to 5,600,000, having an average of 4,666,935. The average of the latter is 5.9 per cent lower than the former. As with the hemoglobin values, all red cell counts for Class 2 cases lie between the highest and lowest Class 1 figures, with the exception of Case 41. This shows that dental conditions such as these do have a tendency to lower both the red cell count and the hemoglobin. The definition of anemia states that these two things must take place. Therefore, we can conclude that dental infection *per se* does produce a mild grade of anemia even in healthy adults. A recent paper by Bryan[6] and one by Adams[7] report an increase in the hemoglobin and the number of red cells after the elimination of dental infection of various types. This is in direct accord with the foregoing findings.

On examination of the structure of the red blood cells in the stained smears, Case 41 was the only one which showed a moderate variation in the size of the red cells (anisocytosis), diminution in the amount of hemoglobin in the individual cells (hypochromasia) and the presence of few cells with slight amounts of blue spongioplasm (polychromatophilia).

Table 9.—Differential Leukocyte Counts

	Class 1			Class 2			General
	Average	Highest	Lowest	Average	Highest	Lowest	Average
Neutrophils	59.5	71.7	47.9	54.7	70.6	48.1	57.1
Lymphocytes	32.1	40.8	21.5	34.6	36.0	25.9	33.4
Monocytes	6.3	11.1	4.5	7.3	10.9	5.4	5.9
Eosinophils	1.9	4.9	0.4	2.1	3.6	0.4	2.0
Basophils	0.7	1.7	0.0	0.9	3.0	0.1	0.8

LEUKOCYTE COUNTS

The leukocyte counts (Table 4) in Class 1 cases average 8,427 cells per cubic millimeter. The range is from 6,950 to 9,850. The average for Class 2 cases is 8,405, a difference of only 21 cells per cubic millimeter between the averages of the two groups. There is a more interesting variation in the dis-

6. Bryan, A. W., et al.: Morphologic Changes in Blood Following the Extraction of Pulpless Teeth, J. A. D. A., 15:1512 (Aug.) 1923.

7. Adams, W. Claude: Significance of the Blood Count in Oral Infection, Dent. Cosmos, 70:314 (March) 1928.

tribution of the counts (Table 5): Class 2 cases are more frequent in the lower and the higher levels, which indicates that both stimulation (producing mild leukocytosis) and suppression (causing mild leukopenia) of the hematopoietic apparatus is in effect. In checking the leukocyte counts, red cell counts and the hemoglobin readings, with the clinical and roentgenographic findings, no apparent correlation was found.

NORMAL DIFFERENTIAL COUNTS

In 1911, Bunting[8] reported a series of twenty-five cases in persons "all apparently in health and none conscious of any illness," in which there were "points of difference" between his findings and "those cited from textbooks," the neutrophil percentages being lower than accepted minimums and the lymphocyte and monocyte values above the accepted maximum values. Since then, his results have been duplicated by others. The figures which are reported below are also in general accord with his findings. This is mentioned because of the prevalent tendency to ascribe diagnostic importance to moderate degrees of lymphocytosis, which, in reality, lie within normal limits.

The differential counts and the study of the structure of the leukocytes was done with the qualitative differential form,[6] which I reported to this Section during the last Session. This form is based upon the functional, regenerative and degenerative changes which take place within the cells. The fact that these changes take place during severe infectious processes such as pneumonia, peritonitis, tuberculosis and septicemia, is well recognized, and this knowledge furnishes a reliable index of the patient's condition when these changes are checked with the clinical symptoms. Increase in round, band, toxic and fragile types of neutrophils; young, lobulated and toxic types of monocytes; leukocytoid forms of lymphocytes, having vacuoles, azure granulation and cytoplasm more basophilic than usual, and unclassed lymphocytes, and the frequent disappearance of the eosinophil during severe cases are recognized by thorough workers in hematology.[9] It was hoped that such

TABLE 10.—NEUTROPHIL LEUKOCYTES

	Class 1			Class 2		
	Average	Highest	Lowest	Average	Highest	Lowest
	59.5	**71.7**	**47.9**	**54.7**	**70.6**	**48.1**
Myelocyte	0.01*	0.2	0.0	0.01	0.2	0.0
Toxic	.....	.....	.....	1 cell	.....	.....
Band	2.3*	6.9	0.0	3.9	9.0	1.1
	(In 95% of cases)			(In 96% of cases)		
Toxic band	0.1*	0.3	0.0	0.4	1.9	0.0
	(In 5% of cases)			(In 52% of cases)		
Segmented	91.9	98.6	76.0	84.9	94.1	73.6
Toxic seg	0.3	3.0	0.0	3.4	11.4	0.0
	(In 84% of cases)			(In 92% of cases)		
Fragile	5.3	19.2	1.4	6.2	13.5	1.9

*All figures represent percentages of the total number of neutrophils.

8. Bunting, C. H.: Normal Differential Leucocyte Count, Am. J. M. Sc., 142:698, 1911.

9. Logefiel, R. C.: Value of Blood-Smear Examination in Medicine and Surgery, Journal-Lancet, 44:617 (Dec. 15) 1924.

an analysis of blood in cases with mild chronic infection, such as dental infection, etc., would reveal findings which could be used to determine the presence and possibly indicate the degree of such infection, where undisturbed major proportions of cells and counts were found during health.

NORMAL QUALITATIVE DIFFERENTIAL COUNT

Neutrophils. — Neutrophils are divided into seven classes: the myelocyte, toxic myelocyte, band, toxic band, segmented, toxic segmented and fragile forms. Single myelocytes were found in six cases, and, in two other cases, two of these were seen. This is in accord with the findings of others. Band neutrophils were found in 96.6 per cent of the neutrophils in Class 1 cases. They range between 6.9 per cent and 0 per cent, averaging 2.3 per cent. Piney[10] states that these cells always form about 4 per cent of the total leukocytes. In 5 per cent of the cases, toxic band forms were seen. They average 0.1 per cent and do not exceed 0.3 per cent of all polymorphonuclears. The segmented forms are the most abundant. They range from 76.0 to 98.6 of all the neutrophils, averaging 91.9 per cent. Toxic segmented neutrophils were found in 84 per cent of the cases. Their portion of the average is 0.3 per cent, lying between 0 and 3.0 per cent. In all cases, fragile or degenerated neutrophils were seen. They average 5.3 per cent and have 19.2 per cent for the higher limit and 1.4 per cent for the lower. The neutrophils form an average of 59.5 per cent of the complete differential, ranging from 47.9 to 71.5 per cent.

Lymphocytes.—The lymphocytes form 32.1 per cent of the Class 1 cases, 21.5 per cent being their lowest value and 40.8 per cent, their highest. These are divided into the micro (small), meso (medium), and macro (large) forms, with a leukocytoid form for each. The first of these (micro) are found to number from 4.5 per cent to 23.2 per cent. They average 23.2 per cent of all the lymphocytes. In 23 per cent of all the cases, leukocytoid microlymphocytes were found. These never exceeded 1.4 per cent and averaged 0.1 per cent of all the lymphocytes. Mesolymphocytes were found in all cases (average, 50.9 per cent; highest, 77.4 per cent; lowest, 0.8 per cent). Leukocytoid mesolymphocytes

TABLE 11.—LYMPHOCYTES

	Class 1			Class 2		
	Average	Highest	Lowest	Average	Highest	Lowest
	32.1	**40.8**	**21.5**	**34.6**	**36.0**	**25.9**
Micro	23.2	48.8	4.5	30.3	48.6	18.7
L. micro	0.1	11.44	0.0	0.2	1.2	0.0
	(In 23% of cases)			(In 24% of cases)		
Meso	50.9	77.4	11.8	32.3	60.7	17.2
L. meso	21.6	54.2	5.8	27.5	46.5	4.9
Macro	0.6	3.5	0.0	0.2	1.1	0.0
	(In 50% of cases)			(In 48% of cases)		
L. macro	1.0	7.1	0.0	1.4	3.4	0.3
	(In 63% of cases)			(In 96% of cases)		
Unclassified	8.4	31.0	1.9	8.1	21.7	2.1

10. Piney, A.: Importance of Hematology in Surgery, Brit. J. Surg., 14:9 (July) 1926.

also were found in all cases and varied from 5.8 per cent to 54.2 per cent (average, 21.6 per cent). Macrolymphocytes were found in 50 per cent of the cases. They constitute an average of 0.6 per cent of the lymphocytes and did not exceed 3.5 per cent of them. Leukocytoid macrolymphocytes, the largest cells in the blood stream, were found in 63 per cent of all the cases. Their highest value was 7.1 per cent; average, 1.0 per cent. The unclassified lymphocytes are those which were either distorted badly or ruptured so that they could not be included with the others. They may form or so-called transitional, form the bulk of the monocytes. The low limit is 20.5 per cent; the average, 55.5 per cent, and the highest, 86.8 per cent, of all the monocytes. In 35 per cent of all Class 1 cases, toxic transitional forms of monocytes occur. The group average is 0.4 per cent; the highest, 8.0 per cent. Lobulated monocytes form 18.3 per cent of the total number. They vary from 1.3 per cent to 61.7 per cent. Of the Class 1 cases, 23 per cent had toxic lobulated forms. They range from 0.4 per cent to 8.0 per cent and constitute 1.5 per cent of the monocytes.

TABLE 12.—MONOCYTES

	Class 1			Class 2		
	Average	Highest	Lowest	Average	Highest	Lowest
	6.3	**11.1**	**4.5**	**7.7**	**10.9**	**5.4**
Young	0.9	5.4	0.0	1.3	5.6	0.0
	(In 35% of cases)			(In 44% of cases)		
Older	24.0	61.1	3.1	26.1	47.6	6.6
Toxic older	0.2	6.0	0.0	1.8	10.2	0.0
	(In 8% of cases)			(In 51% of cases)		
Transitional	55.5	86.8	20.5	52.7	78.8	24.4
Toxic trans	4.4	8.0	0.4	6.0	12.9	1.4
	(In 35% of cases)			(In 96% of cases)		
Lobulated	18.3	61.7	1.3	11.1	39.4	1.7
Toxic lob	1.5	8.0	0.4	6.0	12.9	1.4
	(In 23% of cases)			(In 44% of cases)		

as high as 31.0 per cent of all lymphocytes or as low as 1.9 per cent. Their average is 8.4 per cent.

Monocytes. — The monocytes vary from 4.5 per cent to 11.1 per cent. They form 6.3 per cent of all the white cells. In 35 per cent of the cases, young monocytes were observed. Their average is 0.9 per cent of the monocytes and may go as high as 5.4. The round older monocytes average 24 per cent. They drop to 3.1 and go to 61.1 per cent in another case. In 8 per cent of all cases, toxic variations of the older monocytes were found. The highest reached 6 per cent; the average is 0.2. The indented,

Eosinophils.—The average is 1.9 per cent; the lowest number, 0.4, and the highest, 4.9. No attempt was made to separate them into groups having two, three or four lobes.

Basophils.—These cells did not occur in several cases. When found, they averaged 0.7 per cent and went as high as 1.7.

QUALITATIVE DIFFERENTIAL ANALYSIS OF CLASS 2 CASES (WITH DENTAL INFECTION)

The figures for the five varieties of leukocytes of both groups are quite close together. For example, the average for the neutrophils in Class 1 is 59.5 per

cent, and in Class 2, 54.7. These two average 57.1 per cent. Thus, it can be seen that the neutrophil average for the eighty-five cases is 2.5 per cent higher than Bunting's figure (54.6 per cent) for a similar group. The lymphocyte, monocyte and eosinophil averages for the two classes of cases lie a little over 1 per cent apart. It can be concluded, then, that dental conditions in healthy young men do not produce an appreciable alteration of the five types of white corpuscles. Table 8 can be referred for the detailed findings of the Class 2 cases. There is a more interesting variation between the subdivisions of each variety of leukocyte when comparing the two groups (Tables 8, 9, 10, 11 and 12).

The band neutrophils which occur in 2.4 per cent of Class 1 cases become 4.3 per cent in Class 2 cases. Toxic band forms which were found in 5 per cent of Class 1 cases are found in 52 per cent of Class 2 cases. Toxic segmented forms found in 84 per cent of Class 1 cases are found in 92 per cent of Class 2 cases. Comparing the averages of toxic neutrophils in both classes, we find 0.4 per cent in Class 1 and is compared with 3.8 per cent in Class 2. Fragile neutrophils in Class 1 amount to 5.3 per cent. In Class 2, the figure is 6.2 per cent. Leukocytoid macrolymphocytes are found in 63 per cent of Class 1 cases and in 96 per cent of Class 2 cases. Comparing the general averages of the three types of leukocytoid lymphocytes, 22.7 per cent are found in Group 1 and 29.1 per cent in Group 2. Young monocytes are found in 35 per cent of Class 1 cases and in 44 per cent of Class 2 cases. Toxic older monocytes, found in 8 per cent of Class 1 cases, are found in 51 per cent of Class 2 cases; toxic transitional monocytes, in 35 per cent of Class 1 cases and in 96 per cent of Class 2 cases; toxic lobulated monocytes, in 23 per cent of Class 1 cases and 44 per cent of Class 2 cases. Comparing the averages for young monocytes in those cases which have them, 0.9 per cent in Class 1 is compared with 1.3 per cent in Class 2 cases. The percentages of toxic monocytes as a group occur in 6.1 per cent of Class 1 cases and 13.8 per cent of Class 2 cases. Although there is a slight variation between the eosinophil and basophil figures, the difference is not significant. It may be pointed out here, as with the hemoglobin and red cell counts, that most of the Class 2 values lie between the limits of the Class 1 or normal range. Checking the results of this type of blood cell analysis, no apparent correlation between the extent of change in the character of the blood cells and the type of clinical dental conditions could be established.

These changes which are reported are not characteristic for an individual case with one or more of the clinical findings as noted. The findings bring out group tendencies only. Diagnostic importance should not be given to findings by this method unless the alteration is well outside the normal limits. As noted above, although this is not uniformly characteristic for individual cases, because some cases do not follow the general trend, but merely shows the group tendency, such a tendency cannot be disregarded nor can it be regarded as specific for dental conditions without some consideration of findings from individuals with other types of chronic infection, such as is found in the tonsils, sinuses, bladder, middle ear and other localities. Occasionally, it will probably be extremely useful to make such a detailed analysis of the blood cells as this in cases of uncertain origin. It should suggest the presence of chronic infection.

SUMMARY

The hemoglobin values, erythrocyte counts, leukocyte counts and a morphologic analysis of at least 1,000 leukocytes in each case were studied in sixty cases of individuals who were physically and normally healthy as determined by history, physical examination and dental examination, including roentgenograms of the teeth (full mouth) and vitality tests where indicated. Another group of twenty-five cases comparing with the first, except for the presence of dental conditions, were studied similarily to note what difference, if any, existed between the two groups of findings.

CONCLUSIONS

1. The diagnostic methods at the disposal of the dental clinician frequently reveal no information which might aid in detecting the presence of dental infection.

2. The field of laboratory diagnosis has produced no method which would indicate that the patient is absorbing harmful bacteria and their byproducts at a given time.

3. The literature of dental findings in cases of dental infection shows an interesting variation of opinion.

4. The hemoglobin values in this series average 5 points lower in cases with dental infection than in those without it. With the exception of one case, all Class 2 values lie between the limits of (the normal) Class 1 values.

5. The red cell counts correspond to the difference found in the hemoglobin values, this difference being 5.9 per cent.

6. On the basis of a lowered hemoglobin and lowered red cell count, it can be concluded that dental infection *per se* does produce a very mild grade of anemia in healthy young adults.

7. Case 41 presented a moderate amount of anisocytosis, hypochromasia and polychromatophilia — morphologic evidence of a mild toxic secondary anemia.

8. The distribution of the leukocyte counts showed the values for Class 2 cases to be more frequent in the higher and lower levels (counts).

9. The total differential and leukocyte counts showed practically no variation.

10. Qualitative analysis of the histology of the blood cells showed an increase in numbers of band, toxic, and fragile neutrophils, leukocytoid lymphocytes and young, toxic and lobulated monocytes in the presence of undisturbed major cell proportions. As with the red cell and the hemoglobin values, the difference in the majority of cases lies within the normal range of variation.

11. While there is a definite group tendency, diagnostic importance cannot be given to either the quantitative or the qualitative blood cell analysis in cases with dental infection unless the values exceed the normal limits of variation.

12. The changes noted cannot be considered pathognomonic for dental conditions without consideration of the effect on the hematopoietic system of other pathologic conditions which may be present at the same time.

13. This work has emphasized the value in making blood examinations of the detailed examination of constituent cells.

357 Lowry Building.

DISCUSSION

Weston A. Price, Cleveland, Ohio: Dr. Epstein, do your researches include the older groups in whom there is some evidence of systemic involvement? Have you limited the study to healthy young individuals who have dental infection and show as much change as you have stated here? Might we not expect more marked changes in more advanced

cases of infection? Have you related in your studies the variations in accordance with the degree of infection in the total number of polymorphonuclear and smaller and larger lymphocytes relating to degree of infection?

Dr. Epstein: Considering the fact that I had to go through the health service records and weed out the cases; make the arrangements with patients to come for the full mouth roentgen-ray films and to come back for subsequent examinations; make complete duplicate counts and hemoglobin determinations; take smears, not only one but ten or fifteen, selecting the best, staining and then examining them, I have not had time to go into the older age groups; but the problem as outlined intends to include these. First, we set out to establish the normal variation for each age group. It is only by careful work that we can get figures of value. The next step would be to include older people of both sexes. It becomes a tremendous problem for one man to handle. I presume that later I shall have some facts to answer you with, definitely. As to your second question, Dr. Price, surgeons frequently expect to find much pus when they have a high leukocyte count, with associated neutrophilia. They usually associate the neutrophilia with the presence of pus, and they are frequently disappointed. It is rather the proportion of round, band, segmented or older forms of neutrophils; toxic forms and their toxic variations, and the character of the lymphocytes and monocytes that will give more accurate information than taking the total number of them.

Dr. Price: Do you use the Arneth index?

Dr. Epstein (closing): Arneth's index is quite elaborate. He goes into it in a precise, detailed manner that is not of practical value for routine use because of this. In an examination such as I have presented here, we classify the round, the band and the segmented forms, and, in addition, the functionally altered and toxic variations of the cells. It is simple. It does not take a technician very long to run through 200 cells, and that is all that is done in the usual routine work in a hospital. When we work on an older age group in which the tendency to fight off untoward influences is not so great as it is at the age of the individuals I took for this series, I look for some interesting changes. The findings in such an examination may denote the presence of infection and possibly indicate the degree of such infection by the extent of deviation from the normal range of variation.

Dr. Price: I think we will find a very great difference in the blood picture in an individual who is making a satisfactory and adequate warfare against infection from that in the person who is making an inadequate defense. And I think it will be a measure in a large part of that capacity to fight.

MOUTH INFECTION IN ITS RELATION TO SYSTEMIC DISEASE*

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MOUTH infection, or stomatosis, in its broader sense, includes not only infections of the gums, the peridental membrane and the tooth pulp, but also diseases of the tonsils and sinuses, and the various forms of stomatitis, including the specific infections of syphilis and tuberculosis. William Hunter, of London, in 1900, designated this condition as oral sepsis.[1]

Of this very large subject, I shall deal with only certain phases in which the dental and medical professions are jointly interested.

The two most common types of oral sepsis are pyorrhea alveolaris, or periodontoclasia, and periapical or alveolar

*Read before the Sixth District Dental Society, Macon, Ga., Nov. 13, 1928.

1. Hunter, W. H.: Oral Sepsis, 1901.

abscess. The important relationship of the tonsils to these conditions, I shall mention later.

The relationship of oral sepsis to systemic disease constitutes the most important problem confronting the dental and medical professions today. During the past few years, much valuable research work has been done in this field by both professions; but it is to the dental profession that credit is due for first calling attention to the dangers of oral infection.

The relationship of oral infection to systemic disease attracted the attention of earlier workers in both professions. Benjamin Rush, in 1801, reported the cure of a case of rheumatism by the extraction of a diseased tooth.[2] Miller, in 1889, demonstrated that infections in the mouth may cause constitutional disease.[3]

Even the earliest writers recognized the importance of oral infection. In the Egyptian papyrus Ebers, 1550 B. C., reference is made to gingival disease. Hippocrates, 460 B. C., called attention to diseases of the teeth as a cause of necrosis of the jaw; and Aristotle, 384 B. C., wrote of diseases of the teeth with considerable detail.

A careful physical survey of those who consider themselves in good health will often reveal the presence of serious systemic disease due to focal sepsis in the teeth or elsewhere.

In a given case, the relationship between oral sepsis and systemic disease requires careful investigation by both the dentist and the physician; and such investigation should not only be careful and painstaking, but should also be fortified by definite knowledge of the pathology and clinical manifestations involved.

Both dentist and physician should be familiar with the relationship of focal disease to disease of the heart, blood vessels, kidneys, joints, stomach, gallbladder, glands, appendix and eye, and certain diseases of the blood, the nervous system and the brain, and the pathology involved. They should especially be able to recognize its relationship to hypertensive arteriosclerosis.

Oral sepsis is so commonly found that it is often thought to bear a causal relation to whatever disease the patient may be suffering from, and it may often happen that, without proper understanding and investigation, healthy, useful teeth are needlessly sacrificed.

Pyorrhea is perhaps less important than periapical infection from the standpoint of systemic disease, because, in pyorrhea, the pus usually drains into the mouth; while, in periapical infection, being sealed up, it drains into the blood stream. There are said to be no lymphatics in the alveolar process.[4]

Organisms of low virulence which find their way into the blood stream from a periapical focus, or elsewhere, are either destroyed by leukocytes or the protective substances therein, or maintain themselves long enough in the blood stream to find lodgment where conditions are favorable to their growth, or where there is natural impairment of the circulation, as in the terminal capillaries in the heart valves, kidney glomeruli, gallbladder, or appendix, or in the vasa vasorum of the blood vessel walls, and elsewhere. They may find lodgment where there is accidental impairment of the circulation resulting from trauma,

2. Rucks, W. W.: Systematic Diseases of Focal Origin with Especial Reference to Dental Causes, J. Oklahoma S. M. A., 19:286 (Nov.) 1926.

3. Miller, W. D.: Die Micro-Organism der Mondhole, Leipsic: A. Thieme, 1889, p. 325.

4. Gray's Anatomy, Philadelphia: Lea & Febiger, 1913, p. 1212.

which produces inflammatory stagnation of the blood stream and necrosis or necrobiosis.

Schamaus says, "All circulatory disorders which reduce the blood supply of a part below a certain minimum, and which cannot be compensated for within a certain period, must end in the death of the tissue affected. This is called indirect or circulatory necrosis."[5]

If death of a part takes place slowly, with some of the characteristics of the terminal life processes, it is spoken of as necrobiosis.

Such a condition of circulatory death undoubtedly results, in the vast majority of cases, when a tooth loses its circulation through disease or trauma. The inflammation which results, either acute or chronic, offers a fertile field for the implantation of bacteria which may be circulating in the blood stream.

Haden cultured 915 pulpless teeth and found only 14 per cent sterile.[6]

When a focus of infection exists in another tooth, in a sinus, in the tonsils, in the prostate, in the uterine cervix or elsewhere, and there is a pulpless tooth, it is most certain to become infected through the blood stream and become an associated focus. In many instances, if pulpless teeth are not infected directly through the root canal, they become infected through the blood stream from diseased tonsils; thus, these two conditions are often found coexisting.

Mead says, "There are two sources of nourishment for the tooth; the dentin receives its nourishment through the pulp, and the cementum receives its nourishment through the pericementum. When the pulp of a vital tooth is removed, the dentin becomes dead. The dentin is in contact with only the enamel and the cementum except at the apical opening, and the tooth may still remain functional, owing to the fact that the cementum attaches the tooth to the surrounding tissue and receives its nourishment from the surface and not from the dentin. Where the pulp is lost, the tooth may probably be retained if there is no peridental or periapical pathology."[7]

The artery which supplies both the pulp and the pericementum is the apical artery, which, just before it enters the apical foramen, sends off branches to supply the pericemental membrane, these branches ultimately forming a plexus beneath the cemental and alveolar surfaces of this membrane. The inner surface of this membrane is a reflexion of the periosteum of the alveola, but the two are combined in a single fibrous sheath; on the inner surface, which serves blood to the tooth, are to be found cementoblasts, and on its outer surface, which supplies nourishment to the alveolar bone, are found osteoblasts. (Gray's Anatomy.)

It is unreasonable to presume that when disease or trauma causes death of the pulp, the destructive process is limited to the vessels within the pulp cavity. It is probable, in every instance, that the vessels outside the apical foramen are also affected, and it is very likely, in most instances, that the arterial branches given off to the pericementum just outside the apical foramen are occluded by inflammatory thrombosis or necrosis. It is possible that a certain amount of circulation is regained collaterally, but it is not likely that the life of the tooth is maintained.

5. Schamaus, Hans: Textbook of Pathology and Pathological Anatomy. (Trans. by Thayer, A. E.) Lee Bros. & Co., 1902, p. 51.

6. Haden, R. L.: Pulpless Tooth from a Bacteriologic and Experimental Standpoint, J. A. D. A., 12:918 (Aug.) 1925.

7. Mead, S. V.: Diseases of the Mouth, St. Louis: C. V. Mosby Company, 1928, p. 179.

The production of disease following bacterial invasion is dependent on several factors. As bacteria are not always able to grow in the tissues, invasion often occurs without the production of disease, the organisms being destroyed by leukocytes or the chemical substances in the blood which are antagonistic to their growth, or through being unable to find suitable food.

The factors concerned in the production of bacterial disease are: the virulence of the invading bacterium; the number of invading bacteria; the general and local susceptibility of the host, and the presence of bacterial toxin.

Vaughn says, "Bacteria live and multiply through the activity of their enzymes. Their extra-cellular enzyme splits the pabulum with which they are in contact into proper blocks, and their intra-cellular enzyme fits these blocks into the bacterial molecule. It is plain that a bacterium whose enzyme cannot act on body proteins cannot infect the animal."[8]

Such bacteria grow outside the body. They may be found growing in the mouth and other body cavities, although not be able to grow within the tissues, as they do not find there suitable food.

The presence of bacteria in the tissues does not necessarily mean infection, which occurs only when the bacterium finds the environment suitable and is able to live and multiply.

Small wounds and abrasions of the skin and mucous membrane afford an opportunity for bacterial invasion, which usually occurs, though the wounds heal without showing any evidence of infection. Such wounds are said to heal by primary intention. But if there is much laceration of tissue, a fertile soil may be prepared and infection occur. Infection is always expected to occur in compound comminuted fractures unless great care is used in cleaning the wound and rendering it aseptic.

Osler uses as a simile the parable of the sower: "There are tissue soils in which the bacilli are, in all probability, killed at once; the seed has fallen by the wayside. There are others in which a lodgment is gained and more or less damage done, but finally the day is with the conservative, protecting forces; the seed has fallen upon stony ground. Thirdly, there are tissue soils in which the bacilli grow luxuriantly, and the day is with the invader; the seed has fallen upon good ground."[9]

Streptococci found living in the mouth under purely saprophytic conditions are usually not capable of producing disease; they are nonpathogenic. But when they become implanted in the tissue under favorable conditions, they may adapt themselves to a parasitic existence and thus become capable of producing disease; i. e., they become pathogenic.

Price says, "The strain of streptococcus most frequently found in the mouth is the streptococcus salivarius. It has, ordinarily, a very low disease producing quality. It is the strain most frequently available for infection in caries and pyorrhea pockets."[10]

Rosenow says that "cultures from the buccal mucous membrane and the surface of the tonsils and throat when injected into animals usually did not produce disease, the animals remained well and entirely or relatively free from

8. Vaughn, V. C.: Infection and Immunity, J. A. M. A., 1915, p. 225.

9. Osler, William: Principles and Practice of Medicine, New York: D. Appleton & Company, 1902, p. 271.

10. Price, W. A.: Systemic Expressions of Dental Infections, Ann. Clin. Med., 4:943 (May) 1926.

lesions; whereas, strains from the depth of a focus produced characteristic lesions."[11]

Such comparatively harmless streptococci of the mouth may gain entrance to the healthy pulp through abrasion, erosion or caries and not produce disease.

The virulence, or the invasive power, of bacteria depends, primarily, on the ease with which they can live on body proteins and adapt themselves to environmental conditions within the host.

Price says, "The fact that streptococci are found in all dental lesions of the apical area is probably dependent upon the very remarkable range of adaptability of that organism to environment."[10]

Rosenow has presented evidence indicating mutation between streptococci and pneumococci.[12]

Price has shown that strains of streptococci which required a dilution of 40 per cent solution of formaldehyd, U. S. P., of 1:10,000, in order to permit growth on the medium used, came finally to grow, by suitable transfers, in a concentration of 1:320.[10]

Rosenow has shown that strains of streptococci having a definite elective affinity and retaining it for weeks or months in periapical structures, when aerobic platings were made lost their elective affinity completely.[13]

The loss in virulence of streptococci and other bacteria when cultivated upon artificial mediums, and their increase in virulence on their passage through animals, is well known.

Such organisms as *Bacillus typhosus* and the alpha and beta strains of pneumococci find conditions within the body particularly suitable to their growth, and, when introduced, multiply rapidly and liberate great quantities of toxin, until they cause a powerful tissue reaction, with the production of immune substances in sufficient amount to overcome them. These bacteria are types of high virulence.

There are other invaders, those with which we are concerned at present, of low virulence, which are not sufficiently toxic to arouse a tissue reaction to any great extent, such as the spirochete of syphilis, the tubercule bacillus and certain streptococci of focal disease.

The ability to maintain growth in the human body varies greatly in bacteria, and they may be easily attenuated by unfavorable conditions, such as remaining in prolonged contact with the products of their own metabolism, as may happen in granulomas completely incapsulated in fibrous tissue, which are often sterile.

Bacteria may gain entrance to the normal tooth pulp through decaying dentin and be unable to maintain growth, or be able to maintain growth only for a time; but if the tooth pulp is diseased, conditions for their growth are more favorable.

Hartzell reports that he found vital pulps of forty-one teeth out of 115 to contain growing micro-organisms.[14]

Haden cultured 392 vital teeth, and obtained a growth in 54 per cent; although he says, "Some of the teeth considered as vital were probably pulpless, since all were not tested for vitality, and others had large cavities, and might well have already infected pulps."[6]

11. Rosenow, E. C.: Changing Concepts Concerning Oral Sepsis, J. A. D. A., 14:117 (Jan.) 1927.

12. Rosenow, E. C.: Transmutation within the Streptococcus-Pneumococcus Group, J. Infect. Dis., 14:1 (Jan.) 1914.

13. Rosenow, E. C.: Quoted by Billings, F. T.: Lane Medical Lectures, New York: D. Appleton & Company, 1916, p. 33-47.

14. Henrici, A. T., & Hartzell, T. B.; Bacteriology of the Vital Pulp, J. N. D. A., 7:375 (April) 1920.

When small numbers of bacteria gain entrance to the tissues, they are met by a protective barrier, which disposes of them, if they are not too resistant. In order to produce disease, the quantity introduced must be sufficient to overcome, temporarily, or exhaust, the phagocytic action of the leukocytes and the inhibiting action of the protective substances. If the organism is strongly pathogenic, it may be so thoroughly adapted to conditions of growth in the tissue that the barrier is overcome, and small numbers produce disease. An organism possessing such adaptability is the anthrax bacillus. It is said that a single anthrax bacillus may produce death in a susceptible animal. However, with the organisms of low virulence, with which we are dealing, the barrier above referred to serves to protect the host.

In chronic streptococcic septicemia resulting from focal disease, only at times can streptococci be isolated from the blood stream. The organisms do not maintain themselves within the circulation and are not constantly present there. They are present in the focus, and are, from time to time, discharged into the blood stream. When the individual's protective substances are present in abundance and active; or when the opsonic index for the particular streptococcus is high, the organisms discharged into the blood stream are immediately destroyed. On the other hand, when the protective mechanism is broken down, through some debilitating influence, the streptococci which are discharged into the blood stream from the focus are allowed to multiply unhindered, and an overpowering septicemia, with death of the individual, may result.

Pasteur found that "natural immunity varied greatly from time to time, and could be affected by many debilitating influences. He showed that the marked immunity of chickens to anthrax could be overcome by immersion of the fowl in cold water; and from such lowering of the body temperature, the fowl developed the disease."[15]

If the individual's resistance is only slightly lowered, and the protective mechanism is only weakened, some streptococci may be destroyed, while others, a little more resistant, will be able to maintain themselves for a time in the blood stream, such streptococci ultimately finding lodgment where local conditions are favorable to their growth, and there producing disease.

The phagocytes, or the polymorphonuclear white blood cells, constitute the mobile army of defense against the bacterial invasion, while the fixed cells elaborate substances that are chemically destructive. Usually, both of these forces are concerned in combating bacterial invasion.

The protective chemical substances, natural and acquired, are many: agglutins, antitoxins, bacteriolysins, cytolysins, opsonins, precipitins and others. They are spoken of collectively as defensive proteins.

Billings says: "The natural defenses of the body, due to the bacterial and antitoxic powers of the tissue, blood plasma, and cells, especially the phagocytes, protect the majority of us from the acute infectious diseases. All individuals do not possess an equal degree of natural immunity; some more readily succumb to invading infectious agents. When the animal body is invaded with pathogenic bacteria, the natural defenses are increased by their presence in the tissues and blood. The processes are: first, the phenomenon

15. Pasteur, quoted by Billings, F. T.

of positive chemotaxis with resulting leucocytosis and the accumulation of leucocytes in the areas of infection of the tissues, by the formation of local exudates; liquid, purulent and fibrinoplastic, which may serve as walls of protection against further direct invasion; second, leukocytic phagocytosis with the destruction of the invading bacteria; and third, the formation of protective antibodies in the blood and tissues."[16]

The Lane Lectures on Focal Infection by Frank T. Billings, from which the foregoing is transcribed, should be read by every physician and dentist.

Canfora found that "tetanus spores inoculated under the skin are rapidly distributed throughout the circulation, and if no trauma took place at the point of inoculation, the locally lodged spores may be rapidly destroyed; but if, during this period, there is produced a small wound, blood clot or necrotic area in the body, this may serve as a focus for development, and tetanus may ensue."[17]

Endotoxic bacteria, that is, bacteria which contain their toxin within the bacterial cell, produce harmful effects in two ways: first, by the liberation of their endotoxin, through a process of bacteriolysis, poisoning the individual; and secondly, by inducing inflammation in the tissues and causing proliferation of fixed cells, especially fibroblasts.

The disease which fundamentally is the most devastating of all the chronic diseases is fibrosis. It is responsible for impairment of function and severe crippling of the various organs, and though it is a process of tissue repair, it is ultimately destructive to life. It is spoken of as a process of vegetative or formative repair. It results from, and is the final or reparative stage of, inflammation. This process of fibrosis is engendered and maintained by focal infection.

The lodgment and proliferation of bacteria in the tissue is followed by inflammation; and the effect is much the same as that produced by trauma, blood stasis, perivascular infiltration, impairment of local nutrition, the destruction of more or less cellular tissue and, lastly, the proliferation of fibroblasts, forming the scar tissue. It is this process which is responsible for hardening and loss of elasticity in the blood vessel walls in infectious arteriosclerosis, in pinching and deforming the kidney parenchyma in chronic nephritis and in contracting the heart valves, preventing coaptation of the edges and producing a leak. Its deforming effects are responsible for impairment of function in all the organs of the body. It is, usually, a process quite gradual in its progression, but it is certain and sure in its ultimate destruction of life.

This process is mainly due to invasion of the tissues by bacteria of low virulence, which find their way into the blood stream, most frequently, from the tonsils, sinuses and teeth.

Rosenow says "Clinical observations indicate that localized infection in certain structures, such as the tonsils, teeth and sinuses, are more likely to be associated with systemic effects than those in other structures."[11]

Typhoid fever may greatly damage the blood vessels through *Bacillus typhosus* gaining entrance to the vasa vasorum and finding lodgment in the perivascular space of the blood vessel walls. Virulent pneumococci circulating in the blood stream during an attack of pneumonia may find lodgment in the tissues

16. Billings, F. T.: Focal Infection, Lane Medical Lectures, New York: D. Appleton & Company, 1916, p. 16.

17. Canfora: Centralbl. f. Bacteriol. (Orig.) Vol. 45, 1908.

and set up a process of local inflammation which is followed by fibrosis, and this occurs, to a varied extent, in all the acute infections. But the bacteria responsible for the acute infections do not produce such great and widespread damage, as they are organisms of high virulence, and they either destroy the individual, through the effect of their toxin, or they are themselves destroyed by the aroused protective mechanism.

The life of these bacteria of high virulence in the individual is too short to give them an opportunity to produce such great damage.

It is the bacteria of low virulence, of the streptococcus-pneumococcus group; *Streptococcus viridans*, the gray, the indifferent and the slightly hemolytic, and the spirochete of syphilis which produce great devastation. They are organisms which gain entrance to the blood stream through a primary focus; the chancre of syphilis, a septic condition of the tonsils, teeth and sinuses, and from chronic infections of the prostate, rectum and uterine cervix, and elsewhere.

The process of destruction induced by these organisms of low virulence is progressive. The organisms are not sufficiently toxic to produce a specific protective reaction that is maintained, and they become true parasites, and are, more or less, constantly present in the blood stream.

It is not the part of wisdom to say that an individual is suffering from focal disease when he is stricken with apoplexy, the result of fibrosis of the blood vessels and kidneys caused by focal infection. Nor is it wise to wait until one develops acute articular rheumatism, mitral stenosis or acute appendicitis, before foci of infection are looked for and their eradication considered. These are end-results. The individual can usually be saved the pain, the expense, the loss of time, the danger to his life and the shortening of his days, by the early removal of all possible foci of infection.

Faught says, "I am entirely out of sympathy with those who believe that the removal of foci of infection in an effort to prevent, relieve and cure systemic disease, is overdone. To fail to advise the removal of an infected area, when its removal does not jeopardize the life or future health of the individual, implies either negligence or lack of knowledge and experience. What harm if we do remove a number of teeth or a few tonsils without apparent benefit, if, by so doing, we are able to reduce the incidence of those diseases which now contribute to more than 25 per cent of adult mortality."[18]

Stazunsky says, "It has been stated that Rosenow and Gardner would condemn every pulpless tooth, because they are not tolerated under any circumstances at the Mayo Clinic. It has been explained, however, that patients attending the Clinic are all suffering from general systemic disease and, therefore, every possible focus of disease must be eradicated. These views, however, do not apply when general symptoms are absent."[19]

The serious forms of heart disease, due to focal infection, are usually not discovered until great, irreparable damage has been done. Years elapse before arteriosclerosis reaches the stage where recognition is probable, and the associated kidney changes are not appreciated until com-

18. Faught, F. A.: Focal Infection in Its Relation to Medical Problems; Its Uses and Abuses, Am. J. M. Sc., Philadelphia: Lee & Febiger, Vol. 172, p. 718.

19. Stazunsky, J. A.: Pulpless Teeth in Regard to Their Pathological Sequelae, Dent. Mag., 44:71 (Feb.) 1927. (Transcribed from Guy's Hosp. Gaz.)

pensatory hypertension occurs, or until there is serious impairment of renal function.

A parallel might be drawn between this condition and community welfare. Suppose the purification of the water supply of a community were not considered until typhoid fever breaks out and lives are lost?

C. F. B. Stowell, in discussing a paper by Russell L. Haden, said, in part: "A pulpless tooth is a constant potential menace. It demands watching if you keep it in the jaw, and even if you do watch it with every agent at our present disposal, you cannot be sure of its harmlessness. Extraction of the pulpless tooth is, today, the only sure way of preventing its causing systemic complications." He says further, "The pulpless tooth is a perforated test tube. The culture medium is the organic part of the dentin and the organic odontoblast fibers, which are not removable from the pulp. The warm room is the body itself. Like the medium of Dr. Haden's experimental tube, the oxygen tension varies, decreasing to almost ideal anaerobic condition near the apex. Available medications may attenuate the organism, but they cannot sterilize the dead nonresisting interior of the tooth."[20]

Stazunsky says, "The best possible root canal filling is a vital healthy pulp."[19]

20. Stowell, C. F. B.: Disc. of Haden, R. L.: Value of Dental Radiography in Detecting Chronic Periapical Infection, Illinois M. J., 50:62 (July) 1926.

THE INTRA-ORAL VERSUS THE EXTRA-ORAL METHOD OF RECORDING MANDIBULAR MEASUREMENTS*

By FELIX A. FRENCH, D.D.S., Ottawa, Ont., Canada

IT seems but natural that we should have arrived at the intra-oral registration method after travelling in zigzags for fully two decades. Progress, as a rule, advances by detours to reach its destination.

Balkwill interpreted the function of the maxillomandibular apparatus very ably in 1866, and Christensen, in 1902, introduced the wax-bite method; while Walker designed an articulator incorporating the fundamentals of adjustability as early as 1895.

The findings of these pioneers do not in every way conform to our latest views and requirements yet they closely approach the facts. The speculative guesswork and delusions of dental sciolists distorted the underlying truths of that worthy pioneer trio and made it difficult for able researchers to propagate their interpretations and progress.

And so it happened that the most ridiculous ideas were received and spread by their sponsors and lieutenants.

Most of us present witnessed the confusion that resulted from misinterpretations and distortions of sound fundamental ideas. An endless number of

*Read before the Section on Full Dentures at the Midwinter Clinic of the Chicago Dental Society, Jan. 15, 1929.

theories of mandibular function and a still greater number of articulator designs, each claimed by its sponsor to be the panacea for failures in denture construction, made their debut on the market and in dental literature, and tried for supremacy or popularity, this depending on whether the one or the other was the aim. We may, in retrospect, recognize that the usurpation of authoritative supremacy was attempted by emphasizing theoretical aspects, while the popularity hunter ridiculed or abused theory by subsidizing arbitrary methods, automatic instrumentation and guarantees for increased fees.

These unflattering occurrences cannot be accounted for unless we, as a profession, confess to having, if I may quote from Locke, "swallowed down opinions as silly people do empirics' pills."

This was the situation as I analyze it today, without ill-feeling toward the actors and audiences of yesterday.

In honesty to my confrères and myself, it may be in order to record that this criticism partly applies to me, since only seven years ago, I belonged to the audience and swallowed many a sugar-coated prosthetic pill.

It will spread peace and enhance goodwill, if we blot out our recollections of the controversies which filled the decade before the national meeting at Cleveland, in 1923. What happened at Cleveland was a "consummation devoutly to be wished." It must be credited to the energetic efforts of the chairman of the geometry committee of that organization, Robert R. Gillis.

Dr. Gillis practically forced a just consideration of the Hanau intra-oral registration and check method in the face of the then universally accepted Gysi extra-oral tracing method. Both claimed to obtain practical maxillomandibular measurements for adjusting an adaptable articulator through their methods.

The Gysi extra-oral method is an outgrowth and development of the Balkwill findings, Dr. Gysi having designed elaborate devices for its application. The Hanau intra-oral method makes use of the Christensen wax-bite method and presents a technical refinement as far as it makes definite and accurate application possible. The Gysi and Hanau methods are the only two original developments of mandibular registrations which today still deserve serious consideration as an issue of a possibly unsettled controversy.

It seems that, in some quarters, the Gysi method is still upheld for sentimental and commercial reasons. This attitude is hard to understand, as we must imply from Gysi's own recent writings and from the policies of the commercial backers of his instrumentation that Gysi's confidence in the extra-oral method is shaken and he and his associates are gradually and slowly retreating to adopt the truths evidenced by the recorded facts and clinical demonstrations of Hanau.

The Gysi extra-oral method substantially comprises these three main features:

1. An intra-oral registration of centric relation under equalized pressure (?) with the arrowpoint tracing as a check.
2. The making of condylar tracings with a face-bow like scriber for determining the condylar angles of mandibular excursion to be reproduced in the adaptable articulator.
3. The making of sagittally located arrowpoint tracing to conform with lateral excursions of particularly the incisal guidance.

Hanau pointed out and proved in clinical demonstration in cooperation with Drs. Boyce and Nelson before the

American Dental Association at Cleveland in 1923 that the Gysi extra-oral method is not applicable to denture restorations which rely on a support subjected to "realeff." The word "realeff" is formed by assembling the initial letters of "resilient and like effect," for which it stands.

Hanau's proofs clearly indicate that the Gysi extra-oral method must not be applied to denture construction for which it was developed and recommended by Gysi.

Hanau grants applicability of the Gysi extra-oral method to cases not involving realeff, such as is presented by a set of sound natural dentures.

A consideration of realeff as a factor affecting measurements and the development of a complete method of securing acceptable maxillomandibular registrations applicable to articulator adjustments is an outstanding accomplishment of Hanau.

Hanau's findings and recommendations are not as yet generally accepted in the profession. Holzmann, of Switzerland, and Max Mueller, of Germany, consider the Gysi extra-oral method correct and the Hanau intra-oral method utterly useless. That appears strange in face of the evolution of ideas that Gysi and his followers are undergoing.

The outstanding features of the Hanau intra-oral method are:

1. The consideration of realeff by applying a modified Christensen wax record method enabling the making of zero-pressure records of all jaw relations needed for initial articulator adjustments, and consideration of pressure records for secondary articulator adjustments.

2. A methodical recheck of all mouth records by articulator records.

3. The systematic cooperation of the patient by accurate interpretation of his tactile sense and the physiologic reaction of tissues.

4. Relief of the masticatory surfaces by a correcting grinding technic to accommodate function of the masticatory surfaces under pressure.

5. A simplification of instruments and technic universally applicable to restoration of masticatory surfaces of natural, partly restored and artificial full dentures.

After vain attempts to apply various recommended arbitrary technics and the Gysi extra-oral method successfully, I submitted the Hanau intra-oral method to practical application and I have found its claims substantiated in my daily work during the last few years.

My experiences have decided the issue for me. All that I occasionally apply of the extra-oral technic is the arrowpoint tracing shown by Balkwill and developed in application by Gysi. This arrowpoint tracing remains in my opinion an excellent check on centric jaw relation as a position in conjunction with the intra-oral method of recording and checking a rest relation.

In reviewing the progress of denture prosthesis and also of that special work which relates to the restoration of masticatory surfaces of natural dentures, we will find that the latest renaissance of progressive thinking on this matter occurred at Cleveland when the issue was forced by R. R. Gillis, of Hammond, Ind. We have Dr. Gillis with us today, and I wish to express before this society my appreciation of his attitude, once unappreciated.

IODIZED OIL IN OSTEOMYELITIS OF MANDIBLE: A PRELIMINARY REPORT

By FREDERICK S. BALYEAT, D.D.S., M.D., Los Angeles, Calif.

IN recent years, there has been much work done with iodized oil; chiefly, with the idea of using it only as a diagnostic aid. Lately, some have used it for therapeutic reasons. Some of the conditions in which it is used are: (1) thoracic disease, such as bronchiectasis, lung abscess and empyema; (2) nasal sinus conditions, chiefly the maxillary; (3) spinal lesions (here, however, a special lighter oil is used) and conditions of the genito-urinary tract. It has also been used by a few for the outlining of dental cysts.

Iodized oil as lipiodol was first introduced twenty-five years ago by LaFay, of France. It was originally used in intramuscular injections in cases in which the use of iodin as a therapeutic agent was indicated. It was found that these injections caused large opaque areas to appear in roentgenograms, and that this condition cleared up very slowly. They found only a very few bad effects, such as abscess formation and iodism.

It recently occurred to us that it might aid in the diagnosis of some cases of osteomyelitis of the mandible. We also thought that, on account of its iodin content, it might be of some value in the treatment.

This oil is of high molecular weight, having a specific gravity at 20 C. of from 1.34 to 1.35. It is the oil of the poppy seed with the addition of from 39 to 41 per cent of iodin (0.54 gm. of iodin to the cubic centimeter). It is thick and viscous, and has a peculiar odor, somewhat resembling that of garlic. It is insoluble in water. On exposure to air and sunlight, it decomposes, turning dark brown with the liberation of iodin, which is irritating to the tissues. The iodin is also rapidly set free by alkaline juices, which may cause iodism.

OSTEOMYELITIS OF THE MANDIBLE

Osteomyelitis of the mandible is chiefly caused by trauma, septic and specific infections and the presence of mineral irritants. The symptoms in the more common cases are those caused by trauma, as after extraction or fracture, pain, swelling and general symptoms, such as increased temperature and pulse and loss of appetite. The condition goes on to necrosis, with the typical moth-eaten appearance, as seen in the roentgenogram. We have found that a conservative plan of treatment gives the best results. Drainage should be established early, and the patient should have plenty of rest and an abundance of good food. Tonics should be used and symptomatic treatment given as required. Later on in the disease, after the bone has become separated, a sequestrectomy usually is performed. We have employed iodized oil only in those cases of osteomyelitis following trauma.

TECHNIC

The syringe should be one with tight joints and one that can be used with slight pressure. The oil should be fresh and warmed to body temperature. If

there is an external sinus, it should be stopped up by a tampon soaked in collodion. We have found that it is usually best to inject the oil in the prone position, preferably on the roentgen-ray table. In the actual injection, great care must be used not to get any of the oil in the mouth, as it has a bad taste, and if it is swallowed, there is more danger of iodism, owing to the alkalinity of the intestinal juices. Likewise, none of the oil should escape either internally or externally, as this spoils the details of the roentgenogram. We have used comparatively small amounts, seldom exceeding 5 c.c. This, of course, is subject to variation, depending on how badly the bone is diseased. We also injected the oil at intervals of several weeks because of the slow absorption.

The roentgenograms will show definitely the position of the oil in the diseased bone, but as has been said before, no oil should be allowed in the mouth or on the neck, as this will spoil details.

All of our patients have reacted well to the injections. None have shown any reaction such as fever, chills or local pain. The only complaint that has been made arose when some of the oil escaped into the mouth, and the taste was objected to.

It should be borne in mind, however, that the iodized oil cannot be used in too large amounts as there have been some cases of iodism reported after the use of lipiodol.

CASE REPORTS

CASE 1.—A. M., a white man, aged about 50, of negative history, on Sept. 13, 1927, was severely injured in an auto accident, suffering cerebral concussion, multiple fractures of the mandible and fracture of one of the clavicles.

Examination of the mouth, which showed poor dental hygiene, revealed an open fracture in the region of the left lower cuspid, with considerable mobility and displacement. There was pus coming from the fracture line in the mouth. There was also an acute abscess in the submental region. The roentgenograms revealed also an extra-articular left condyle fracture.

The acute abscess was opened and drained. The lower jaw was immobilized by interdental wiring for about six weeks. All during this time, there was a purulent discharge externally and internally, and several bone spicules came out. When the wires were removed, there was no union of the anterior fracture, and we again immobilized the jaw. In November we gave the first lipiodol injection, with no reaction. This was followed in a short time by a second injection, after which the purulent discharge became much less and the roentgenograms showed that the osteomyelitis was localized adjacent to the fracture line. A third injection was given, December, 1927, after which there was no more drainage.

Laboratory work done at various times showed that the blood Wassermann reaction was negative. The urine showed a few pus and epithelial cells. Blood chemistry revealed nonprotein nitrogen, 33; sugar, 103. A blood count showed: leukocytes, 8,900, erythrocytes, 4,100,000, and hemoglobin, 60 per cent.

We tried immobilization, removal of loose teeth, etc., to no avail. In July, open operation was performed with bony union resulting.

CASE 2.—C. H., a colored woman, aged about 35, of negative history, had a lower right molar extracted about Dec. 1, 1927. Jan. 29, 1928, she came to the Los Angeles General Hospital oral surgery clinic with marked osteomyelitis of the right side of the mandible.

Examination of the mouth revealed much pus coming from the lower molar sockets and poor dental hygiene. There were crepitus and mobility in this region and there was considerable swelling in the right submaxillary region, several sinuses discharging pus.

The blood Wassermann reaction was negative. The urine showed a few casts and epithelial cells.

The patient was given conservative treatment until March 28, when lipiodol was injected, revealing an extensive osteomyelitis and a pathologic fracture. No reaction fol-

lowed the injection. A week later the discharge was some less. The patient left the hospital shortly after and did not return for further treatment.

CASE 3.—Wm. McK., a white man, aged about 43, of negative history, July 1 to 10, 1928, had toothache in the lower molars, accompanied by some external swelling. July 10 the left lower first and second molars were extracted under procain anesthesia. The following day, the face swelled considerably and there was great pain. July 13, the patient entered the hospital. The face and submaxillary region were greatly swollen; the temperature ranged from 102 to 104 F.; the pulse was rapid and there was considerable jaundice. The patient became toxic, and we opened the swollen area, under nitrous oxid-oxygen anesthesia. Considerable pus was obtained. The laboratory reported *Staphylococcus albus* present.

The laboratory examination at this time revealed: white count, 14,000; blood Wassermann reaction, negative; blood chemistry: sugar, 144; nonprotein nitrogen, 42, and icteric index, 40.

July 25, the patient was clinically much improved, but there was still a profuse foul discharge. July 27, Vincent's organisms were found, and the patient was sent to the contagious building for treatment. He returned August 5, and roentgenograms taken at this time revealed a very extensive osteomyelitis of the ramus and body of the mandible. From this time until October 15, there was some rise in temperature and considerable purulent discharge. October 15 lipiodol was injected. One week later, there was less discharge. A second injection has been given, with some improvement.

SUMMARY

It is realized that no conclusion can be drawn regarding treatment of this disease from so few cases. It is hoped that this work will act as a stimulus to others in this field in the finding of some agent that will materially aid in the treatment of this condition. From the results of this investigation, it is believed that possibly iodized oil may have some place in the therapeutics of osteomyelitis of the mandible.

1136 West Sixth Street.

THE PERSONAL FACTORS IN THE MAKING OF A SUCCESSFUL DENTAL ASSISTANT*

By MARIE E. STEVENS, Newark, N. J.

I HOPE that my talk will not be misconstrued as a preachment on the morals of behavior in business; neither do I wish to start with an apology. I am not trying to lecture or read a thesis, but merely to state a few thoughts and observations that stand out most vividly on my mental horizon, and I pass them on to you for your acceptance or rejection.

In our profession, one must possess intelligence and the necessary attributes thereto, which we may call the four disciples: tact, poise, dignity and observation. With all this, if we do not have a natural aptness for our chosen work or, in other words, "personality for the job," we certainly are a square peg in a round hole. Which reminds me of the story told of the little fellow who, left in charge of his tiny brother, called out, "Mother, won't you please speak to baby. He's sitting on the flypaper and there's a lot of flies waiting to get on." The moral of the story is, perhaps we are seated where we do not belong. Possibly,

*Read at the first annual meeting of the Dental Assistants Association of Northern New Jersey, May 17, 1928.

we would make more efficient dentists than just assistants. I'll leave that for you to decide. The great thing in this world, I find, is not so much where we stand as in what direction we are moving. To plagiarize the psychologist, if we are not the "type," it is to our advantage to step in line or step out; for if we don't, we soon find old mother lethargy nursing us with the milk of mental stagnation.

The question is, what is personality? The more I contemplate an explanation, the more I realize that it cannot be measured by a plumb line and rule. You cannot by any flight of the imagination paint a word picture to portray its full significance and meaning, that magic word, personality. Yes, any dictionary will give you some idea. But further investigation seems like chasing dead hopes to the grave yard of your dreams. There is no definition. Personality represents a living, throbbing, pulsating, mental and physical something that is better described as a "spiritual quality" and is felt more than it is seen. Personality is so much more tremendous a thing in its heights and depths than verbal explanation or any transcript of it can be. It is the mirrored reflection that comes to you from a brain that is possibly capable of great things. It is the perfume of the mind distilled from the essence of that divine flower that intoxicates all those with whom it comes in contact. Personality is a broken-off piece of your spiritual estate. It is an inspired creative power given to us as a noble gift and its revelation we see and understand as graciousness of speech, charm of manner, distinction of bearing, accomplishments, character and a noble understanding of human kind.

To practice poise as mere mental gymnastics is ridiculous. Some there are who think poise and dignity are a state of emotional starchiness that precedes a bluff. But if we are not overcome with our own importance, we will learn that poise and dignity are not necessarily ours by inherent right, but are acquired only by cultural growth, the result of stupendous reading of classic and technical essays and books; and the association of people higher in station of life than ourselves. Diligence and a thirst for knowledge with a tenacity and a will to search and acquire it—this gives one poise and dignity. Remember the quotation, "Knowledge is wise that it knows so much, Wisdom is humble that it knows no more."

To speak fluently is a talent possessed by few, but to be a good listener is an art. Know when to talk and when silence is golden; also, there are moments when your departure from the room is in order. It is well not to delay leaving the operating room when you think your absence would we appreciated. Do not stand or sit there like a ship in dry dock waiting to be launched. Have a bearing that is serene but resolute, and commanding the respect of those in a higher and lower station in life than yourself. I do not mean to suggest one should parade as a dignitary of exalted rank or as a paragon of virtue with an air of "keep off the grass," but your carriage and deportment characterizes the difference between familiarity and your desire to be friendly. Try to be graceful and cultivate a low well-modulated voice, for remember the voice is the index of the soul. Be cautious and not insincere with your politeness, for it takes nothing short of a genius to hand out counterfeit diplomacy and not be found out. It is well, when talking on the telephone, not to say "I don't get you" when you really mean you fail to understand or you have difficulty in hearing. Be polite, but as brief as is compat-

ible with the circumstances. Long telephone conversations are wearisome. In your explanation to a patient about his next appointment or the proper technic to employ in cleaning the teeth, do not close your conversation by asking, "Do you understand?" By doing so, you infer that he is devoid of intelligence and slow to understand. Observation on your part will quickly tell you whether another explanation is necessary. Do not explain in a way to suggest that he is a perfect vacuum mentally and you are at the heights of great genius. I know it tries one's patience to the breaking point sometimes to try to impart a simple idea to some people. It reminds one of the saying of Conklin, the scientist, "that wooden legs are not inherited, but wooden heads may be." Impress the patient with his aptitude to understand and apologize for your lack of mental agility in imparting quickly and clearly the subject at hand.

It is well not to argue with obstreperous patients. The more clamorous and noisy they become, the more serene you should be. Converse but little, and then in tones lower and milder than has been your custom. They will soon feel ashamed of themselves and calm down. Keep your ray of reason; it is your only guiding star. By your diplomacy, you have dissipated the mental squall of the patient and likewise the embarrassment of putting your employer out of key for the balance of the day.

Do not forget that an intelligent knowledge of the mechanical side of dentistry and its skilful application is of weighty consequence, for it adds to your ability as an able dental assistant. Some there are who may advance the suggestion that a knowledge of mechanics is nonessential as far as they are concerned. Though it may be relatively true that, in some offices, a mechanical knowledge is not of every-day use, there may be times at least when such a knowledge would aid you to better understand or estimate a piece of workmanship just received from the dental laboratory. You may notice slight defects that could be brought to the attention of the dentist at once. You may be able to save an hour or possibly two hours of valuable time, as possibly the dentist may be so engrossed in other duties requiring his attention that he will be unable to inspect the laboratory work at the time of delivery. Again, the position may be reversed; one may be well qualified in the art of mechanical dentistry, but lacking in other prime requisites in the making of an efficient dental assistant. I refer again to personality, tact, diplomacy and general efficiency. They all must co-mingle or one cannot be an all-round able assistant, as the word implies.

Industry, economy, good health, good cheer—these things all spell efficiency and the lack of them spells inefficiency. Unquestionably, the first step toward efficiency is self-respect, which assures respect for the feelings of others. Unless your mind is functioning properly, it will not intelligently transmit energy. Remember: efficiency turns on the ability to utilize to the best account the passing moments.

A STATEMENT FROM THE BUREAU OF STANDARDS IN RELATION TO THE UTILIZATION OF THE NAME OF THE BUREAU IN ADVERTISING

It has long been the policy of the Bureau of Standards to discountenance the use of its name in connection with advertising in which it is stated or suggested that the Bureau approves of or recommends any commercial product or in which it is stated on the alleged authority of the Bureau that one product is better than others. This policy has been declared and attention directed to it on many occasions; it is believed that it is well understood by industry as a whole and has the approval of industry.

As a matter of fact, there is little or no opportunity for any such statement truthfully to be made on the authority of the Bureau since the Bureau takes meticulous care to avoid statements in reports, or otherwise, to the effect that a product is recommended or generally approved; and only by inference and the exercise of independent judgment is a reader able to conclude from any statement emanating from the Bureau that one product is better than other similar products. In other words, as far as the Bureau is concerned, the buyer must decide for himself what particular brand of goods it is best for him to purchase; he cannot rely on the Bureau to make this decision for him.

Very little consideration should be necessary to convince one that this policy is the only possible one that the Bureau of Standards can reasonably adopt. It is not a function of the Bureau to interfere in commercial transactions by extolling one brand over others. The Bureau makes many tests and researches for the advancement of American industry by ascertaining how better products can be more efficiently made, and by recommending satisfactory standards for materials. General results so obtained are open to all; and through the use of such information, better materials and products are made available to the general public. In order to enable the Bureau to proceed freely with these investigations, it should not be hampered by being drawn into controversies which are bound to result when a concern endeavors to capitalize the Bureau's work, not by taking legitimate advantage of the opportunity offered to improve its product, but by advertising its particular product in the manner described above. Experience has demonstrated that the only way in which complications can be avoided is to discourage the use of the name of the Bureau in such a way. Splendid cooperation has been afforded by industry along these lines and there usually is no cause for complaint. Perhaps it is inevitable that the policy will in some instances be violated. Sometimes, doubtless, it is unwittingly transgressed. There is, in most cases, no difficulty in having the objectionable matter withdrawn when the facts are pointed out. Were the situation otherwise, it would be inevitable that the Bureau would have to take precautions which would eventually greatly circumscribe its work.

In view of the fact that practically all manufacturers abide by the wishes of the Bureau, the disregard of them by

one manufacturer, when it occurs, is all the more important, since it makes it appear that he alone has succeeded in obtaining the approval of the Bureau in relation to the type of material which he advertises. This character of advertising is therefore eminently unfair to all the competitors in that field. Even when the violation has been an innocent one, it seems that there devolves on the Bureau a duty to give such publicity as it may to the fact that the objectionable advertising is unauthorized. This is for two reasons: first, to give additional publicity to and emphasize our policy and thus minimize the probability of frequent recurrences of similar instances; and, second, to prevent an injustice to competing manufacturers in the particular case in question since they may be just as surely injured whether or not the advertising was published in good faith.

Some time ago, the American Dental Association and the Bureau of Standards concluded an agreement for and commenced a cooperative research into dental materials, the results of which should be of very great value to the public. In accordance with the plan, the American Dental Association appropriated a sum of money to be expended in this work and employed a research associate, Dr. N. O. Taylor, to be stationed at the Bureau of Standards, to carry on the work. One of the first investigations undertaken was a survey of amalgam alloys. The Bureau of Standards believes that the investigation made by Dr. Taylor in this connection is an excellent piece of work. He submitted his report in due course to the Research Commission of the American Dental Association. The report was approved by this Commission and by its direction was presented by Dr. Taylor at the annual session of the American Dental Association in Minneapolis. It first appeared in printed form in the April, 1929, issue of THE JOURNAL.

Now had this report been made by the Bureau of Standards in the normal course of its work, it will be clear from what has been said above that the names of manufacturers whose products were included in the investigation, as well as the trade names of the products themselves, would necessarily have been excluded. This course has been followed in prior studies of dental materials by the Bureau. The American Dental Association was not, of course, bound by these restrictions, and since there could be no doubt that the report would be of more constructive value to the dental profession if the names of the products investigated were to be included, they were so included.

The Bureau of Standards considers that the American Dental Association acted in an entirely proper manner in disclosing to its members at its meeting and in its journal the work accomplished by the scientist employed by it and paid from its own funds for the advancement of the dental profession. However, it will be clear from the above that the Bureau of Standards, on its part, should not be put in the position of accepting the responsibility for the publication of the report. It would appear that should any manufacturer of dental materials desire to advertise the results obtained on his own materials, he should give credit to the American Dental Association, and, perhaps, seek the approval of that association for his advertising copy.

It has been brought to the attention of the Bureau that one firm manufacturing dental materials, apparently in haste to utilize the report for its own benefit, has published an advertisement which is highly objectionable to the Bureau of Standards. In this advertisement,

the name of the Bureau is prominently and frequently displayed in connection with the utilization of some of the results contained in the report. In fact, the report is attributed to the Bureau of Standards in toto and the American Dental Association is entirely ignored. Thus, we have a case which falls squarely within the limits of the class of advertising which the Bureau strongly feels should be proscribed.

The firm in question now declares that it was not known to them that their advertising was of a type which was objectionable to the Bureau, that it was presented in good faith and that it will discontinue this character of advertising in the future. This may well be the case. The Bureau readily accepts the disclaimer of the firm in question at its face value. But it has been suggested above that by the printing of an advertisement in a periodical the damage in any particular case is done, unless the attention of the reader is thereafter directed to the true facts; otherwise, the advertisement stands unimpugned as the endorsement of the product in question by the Bureau of Standards. The Bureau apprehends that it cannot allow this impression to stand unchallenged.

In general, the Bureau of Standards desires to bring forcefully to the attention of the dental profession the following facts: When an advertisement is encountered in which it is said that the Bureau of Standards approves or recommends a dental material, or declares one material to be superior to other materials, it can be taken for granted that the statement is not a true one. In the particular case at hand, the dental profession is urged to regard the results given in the report discussed, as the results obtained and published by its own association in the development of a commendable program designed to put scientific facts about dental restorations at the disposal of its members and thus to enable them better to serve the public. It is for the purpose of again emphasizing these facts and clearing up misunderstanding which would otherwise inevitably continue to exist that this article has been prepared and offered for publication to those journals known to have carried the advertisement in question in their pages.

DEPARTMENT OF COMMERCE,
BUREAU OF STANDARDS,
Washington

The Journal of the American Dental Association

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C. N. JOHNSON, Editor

Published by the American Dental Association

All expressions of opinions and all statements of supposed facts are published on the authority of the writer over whose signature they appear, and are not to be regarded as expressing the views of the American Dental Association, unless such statements or opinions have been adopted by the Association.

Communications intended for publication in the scientific or literary pages of THE JOURNAL should be addressed to the Editor. All advertising or business matters should be sent to the Business Manager.

Subscription is included in the annual membership dues. To nonmembers living in the United States or its possessions, the price is $2.50 a year in advance; Canada, $3.00; Australia, $4.00; other countries, $3.25. All foreign subscriptions are to be paid in United States money.

Editorials

JUST BETWEEN OURSELVES

It is remarkable to note the better understanding that is all the while developing on the part of most contributors to dental magazines in the preparation of their manuscripts for publication. The increasing amount of periodical material renders it very important that those who prepare papers for journals shall have an intelligent conception as to how it should be done, and for the most part we find this to be the case.

But there are certain angles to this matter of publication that authors should always consider, and it does no harm to offer an occasional reminder on the most acceptable manner of preparing manuscript. It seems hardly necessary to state that copy should be typewritten. Printers are usually marvelous in their ability to decipher pen-written copy, but the general use of the typewriter today should make it unnecessary for them to spend time in making out doubtful chirography.

Also this should be borne in mind: the typewriting must be of such a character that it will readily permit of editing, because, peculiarly enough, it is a rare paper that does not call for a certain amount of editing, perfect as it may be in a general way. The original instead of a carbon copy should be turned in for publication, and the lines should be double spaced. It is almost impossible to edit single spaced type, and a carbon copy invariably leads to more or less blurring, which makes the typesetting extremely difficult for the linotype man.

Speaking of the linotype brings up another very important matter. The use of the linotype, marvelous as it is, forces the necessity for the manuscript to be prepared accurately before it is sent to the printer so that there shall be as few changes as possible. In the old days when the type was set separately by hand, an alteration in a word or letter was not a very serious matter, but the linotype has revolutionized all this. Now if there is the slightest change to be made in the proof, it necessitates the resetting of a whole line, and occasionally if the length of a word is materially changed, it may mean the resetting of several lines. Therefore, the item of alterations in every printing office soon runs into a big expense—an expense which could be minimized almost to the vanishing point by more care in the preparation of manuscript.

This leads to another consideration which is seldom recognized by authors when they read proof of their articles. Ignoring the fact that their copy should have been as they wanted it when it was submitted for publication, they frequently make such sad havoc of their proof that, in some instances, the entire article might almost as well be set over again. This is a needless expense which should never be forced on a publication. Of course, it is inevitable that mistakes will creep in occasionally which are not at all the fault of the authors, and these are always corrected cheerfully, and there is the other consideration that, in the event of a long time elapsing between the reading of a paper and its appearance in proof, an author may logically have changed his mind about a certain theory and wish to state it differently; a privilege which should always be granted him. (In this connection, the long delay in the publication

of some papers cannot always be laid at the editorial door, an instance in hand of which is the fact that a paper read in December, 1927, was finally turned in to the Editor in January, 1929, the delay being the fault of no one.)

Speaking of proof recalls the fact that occasional resentment is expressed by authors when they return their proofs if the editorial staff has in any way ventured to alter their phraseology or nomenclature. A letter is likely to accompany the returned proof with the statement: "I want this article to appear just as I have written it, word for word." It is, of course, perfectly natural for an author to take this position. His name is attached to that article and it seems reasonable for him to request that he be permitted to express himself as he wishes.

But if he only knew! If he only knew how perfectly ridiculous he would sometimes make himself if given full reign, he would seldom be so insistent. The truth is that no alteration is ever made in editing a manuscript for which there is not a very excellent reason, and most writers are grateful for the changes. If every article that comes to an editorial desk were published just as it is, the magazine would be filled with a motley lot of material, and it would not be long before the thinking men of the profession would be asking the editor what was the matter with him. This is so generally recognized that, very long ago, it became an established rule of publication that the decision as to what shall go in a magazine and the manner of its preparation shall be left entirely in the hands of the editorial staff. If an author does not wish to conform to the usage of the magazine in such matters as nomenclature and construction, he has the one alternative of keeping his material out of that publication. As long as he elects to publish in any given magazine, he must conform—gracefully or otherwise—to the rules in vogue in that editorial office. This to some may seem arbitrary, but it is the only way whereby a periodical can be brought out with any kind of uniformity or system.

At the same time, it should be said that an editor is always glad to receive constructive suggestions from his contributors and readers, and is pleased to adopt them if they appeal to his judgment

and fit into his policy. Lack of understanding is at the bottom of most of our difficulties in life, and this question is no exception to the rule. If the average contributor could visit the editorial office and look into the detail of bringing out a magazine, he would be more charitable of the editorial prerogative and more considerate of the necessary rules that govern it.

COOPERATION

In the affairs of life, cooperation is the essence of success. No great enterprise is ever brought to a favorable fruition without cooperation between the individuals of various groups. In dentistry, cooperation is needed between the general practitioner and the different specialists. Nothing but this cooperation will ever bring to patients the best service, and this is becoming so well recognized that general practitioners are more and more inclined to welcome specialists to their ranks.

It is axiomatic that the interests of the dental trade associations and the interests of organized dentistry are so closely interwoven that, to gain the best results for all, there must be the most cordial cooperation between the two activities. In recent years, this has been progressing in a very encouraging way, and a better understanding is developing all around. The trade interests acknowledge that they cannot get along without the profession, and conversely, the profession recognizes that they cannot get along without the manufacturers.

Therefore, it is only the part of wisdom that each shall help the other in every legitimate way. In our own Association, we owe much to the manufacturers. They cooperate in our activities to the extent of their ability. We provide exhibit space for them at our meetings and they contribute to the expense of conducting the meetings by paying for this space. We should endeavor to see that they are treated fairly in all these arrangements. Not that we shall accept dictation from them as to how our meetings shall be conducted, even if they attempted to dictate, which they never have, but that we shall extend the right hand of fellowship to them in all

those situations in which our mutual interests overlap, and that we shall consult with them freely whenever the welfare of both is at stake.

In the publication of THE JOURNAL, they are offered a medium for advertising their products to the profession—one of the very best mediums for this purpose—and, on their part, they contribute to the excellence of THE JOURNAL by paying for these advertisements and thus making possible the expenditure of more money on the scientific features of the periodical. In every way, each activity is helpful to the other, and they should work together in a combined effort to advance dentistry to a point where it can give the best service to the people.

In line with this very idea of mutual helpfulness, the members of our organization, who constitute the greatest bulk of our readers, should always aim to patronize our advertisers, who are so largely instrumental in making it possible to issue a magazine of this character for the profession. The aim of our advertising censors is to admit only reliable products to the advertising pages, and the lines are continually being more closely drawn, so that the materials advertised are, as nearly as can be judged, of a character to inspire confidence on the part of the purchaser. When materials of a certain type must be purchased, it is only logical to suggest that they be selected from those advertised in THE JOURNAL, which is after all only a legitimate form of cooperation.

Mutual helpfulness is the keynote of success in every activity of life, and when all the inhabitants of the world realize this, we shall have a better world.

ADEQUATE CLINICAL EVIDENCE

The movement for reform in the administration of drugs by the dentist carried on in the pages of THE JOURNAL for over a year has had as its object the rationalization of dental medication, and a realization on the part of the profession of the character of some of these proprietaries. Among the several points stressed were: The formula, qualitative and quantitative, must be readily available to

the profession; and the drug or drug preparation should produce the desired or claimed effects.

It is not necessary to dwell at any length on the first of these two requirements. Unless the composition of a drug or mixture thereof is known, the evident relation between the administration of a drug and the course of disease cannot be interpreted. Indeed, it is not too much to say that the practice of medicine in this country owes a great deal to the insistence of the leaders of therapeutic thought on this point. Without such a fundamental knowledge, the primary relations of cause and effect, without which one cannot presume to be scientific, are lost.

But granting that the formula of a drug or mixture thereof is known, how then is adequate clinical evidence obtainable? This fundamental problem has been and is still a source of discussion among many able men. Yet, in spite of wide divergences on the finer points involved, there is no lack of agreement on the fundamental aspects of the problem. On this point, Prof. Torald Sollmann,[1] of Western Reserve University, expressed himself in the following vein:

> When commercial firms claim to base their conclusions on clinical reports, the profession has a right to expect that these reports should be submitted to competent and independent review. When such reports are kept secret, it is impossible for any one to decide what proportion of them are trustworthy, and what proportion thoughtless, incompetent or accommodating. However, if this were done, it is quite possible that such firms would find more difficulty in obtaining the reports. Those who collaborate should realize frankly that under present conditions they are collaborating not so much in obtaining the scientific value, but rather in establishing the commercial value of the article.

In this short paragraph, Professor Sollmann goes to the crux of one of the worst evils attending the introduction and maintenance of many new drugs and proprietaries of unproven value. Those members of the profession who either for accommodation or desire to be rid of the importunities of a representative of some concern, ill-advisedly yield, giving a so-called record of clinical evidence or testimonial, help to increase the evil the profession must be rid of.

1. J. A. M. A., 69: 198, 1917.

But, to consider clinical reports of the better sort, this is said, "Often the best type of clinical reports—those in which the observations are directed to the significant events and not to mere side lines; and in which the significant events are correctly and adequately reported—generally lack one important essential, namely, an adequate control of the natural course of the disease." Since this cannot be controlled directly, it must be compensated indirectly. For this purpose, there are available two methods:

The first is the statistical method, in which alternate patients receive or do not receive the treatment. This method can be of value only when a large number of patients are available. Even then, its value is limited or doubtful because it cannot take sufficient account of the individuality of cases.

The second method consists in the attempt to distinguish unknown preparations by their effects—a method that might be called the "comparative method" or the "blind test."

In this, the patient, or a series of patients, is given the preparation which is to be tested, and another preparation which is inactive, and the observer aims to distinguish the two preparations from their effects on the patients. Surely, if the drug has any action at all, it will be possible to select correctly in a decided majority of the administrations.

It would not be amiss to point out that unless the practitioner can satisfy himself that these requirements have been met before administering a drug, he is not practicing the therapeutic art according to the best thought. Many practitioners, doubtless, would be more hesitant in giving "clinical reports" of various materials if the giving depended on satisfying these criteria.

The surest road to progress is a healthy skepticism, but with an eye for essential truths that make for progress. Dentistry is now in a renaissance, and its devotees will more and more insist on what has been termed adequate clinical evidence prior to drug administration to their own patients, to the advantage of the profession and those dependent on them for expert advice.

ROBIN ADAIR, B.S., D.D.S., M.D.
(1878-1929)

By SHEPPARD W. FOSTER, Atlanta, Ga.

The dental profession of America was shocked to learn of the untimely passing of Dr. and Mrs. Robin Adair, of Atlanta, Ga., who met their death in an automobile accident, April 26, 1929.

Dr. Adair, with his wife and two of their children, was motoring to St. Simon's Island on the southeast Georgia Atlantic Coast, to spend the week-end, and in making the descent of a long hill, 75 miles out of Atlanta, and passing a heavy truck, one of the tires of the car, a heavy sedan, blew out and the car turned turtle, catching both Dr. and Mrs. Adair under its weight and crushing them to death instantly. In some apparently miraculous manner, the rear doors opened and the two children, Frances and Benjamin, were both thrown out of the car, escaping with minor injuries.

Dr. Adair was born in Gainesville, Ga., February 27, 1878. After finishing high school in Gainesville, he entered the University of Georgia, from which institution he received the B. S. degree in 1899. He entered the Southern Dental College of Atlanta, October, 1899, and from this institution he received the degree of D.D.S., in 1902. After graduating in dentistry, he entered the Atlanta College of Physicians and Surgeons (now Emory University School of Medicine), graduating as a doctor of medicine in 1904.

Dr. Adair specialized in periodontia. His contributions to this field of dental literature have been widely published. He was a pioneer in the oral hygiene movement and, as a result of his deep interest in this subject, his alma mater was induced to create a chair of oral hygiene and pyorrhea alveolaris. The records show that this was the first chair thus designated and established in any dental school. Dr. Adair was elected professor of this branch, a position which he successfully filled until he could, with confidence, turn it over to his successor.

Dr. Adair contributed to dentistry a most valuable book entitled "Practical Oral Hygiene, Prophylaxis and Pyorrhea Alveolaris." He collected one of the most complete dental libraries in the South, a greater portion of which he donated some years ago to the Fulton County Medical Society.

Dr. Adair is accredited with being the first dentist in the south to advocate the dental nurse. He was so impressed with the growing need in this field of health service that in 1928 he organized the Atlanta School of Oral Hygiene. The first class, consisting of twenty-two young women, were preparing for their final examinations at the time of his unfortunate death.

He was vice president of the American Academy of Periodontology and was chairman of the Section on Mouth Hygiene for the coming meeting of the American Dental Association, in Washington, D. C., for which he had already prepared a splendid program.

Dr. Adair was a consistent member of the Baptist Church for years. He was a member of the Psi Omega and Omicron Kappa Upsilon dental fraternities.

Outside his profession, his life was devoted to his family. His happiest hours were spent in the seclusion of his home or while on recreation trips, such as the one on which he had started April 26.

Mrs. Adair was formerly Miss Leonora Ragland, of Atlanta. She was a highly talented, lovable woman and a most devoted wife and mother.

Dr. Adair was the only son of Dr. R. B. Adair, who survives him and who, in his eightieth year, is still actively engaged in practice.

Four children survive: Frances Aline, aged 16, Robin, Jr., 14, Junius Millard, 13, and Benjamin Victory, 10.

In the death of Dr. Robin Adair, dentistry has been robbed of one of its most valuable representatives. His enthusiasm was an inspiration to those whom he drew around him. There is a place made vacant which cannot be filled.

CLINTON T. BROCKETT, D.D.S.

(1850-1929)

By SHEPPARD W. FOSTER, Atlanta, Ga.

Clinton T. Brockett, of Atlanta, Ga., died at his residence, 159 Fifth Street, N.E., April 30, 1929.

Dr. Brockett was born in Alexandria, Va., Sept. 8, 1850. After completing his education at the Academy of Alexandria, he entered the Baltimore College of Dental Surgery, from which he graduated in 1870. He practiced his profession in Baltimore until 1878, at which time he removed to Atlanta, where he was engaged in continuous practice until within a few months of his death.

Dr. Brockett was a typical southern aristocrat. His life exemplified the traditions of his birthplace. His practice was lucrative and his clientele was of a select character. He was beloved by many and well spoken of by all. He eschewed any publicity for his acts of kindness, though his good and charitable deeds were known to be many.

He was a consistent member of the local, state and national dental associations.

Dr. Brockett was for many years a member of All Saints Episcopal Church, and for the past twelve years was one of the vestrymen.

He is survived by one sister, Mrs. Emma Isherwood, of Washington Grove, Md., and three grandchildren, Thornton, Brockett and Betty Comer.

Though death has taken him away,
Who cheered us with his friendly smile,
His memory will always stay;
Only his voice is stilled a while.

JAMES HARRISON PROTHERO, D.D.S., F.A.C.D.

(1862-1929)

James Harrison Prothero was born in Albany, Ill., March 1, 1862. His death occurred, Monday, April 8, 1929, at Wesley Memorial Hospital in Chicago, after a four-day illness, from pneumonia.

Dr. Prothero received his preliminary education in the public schools in Kansas, and he afterwards attended the state agricultural college in Manhattan. In the fall of 1885, he entered the Missouri Dental College, now the Washington University School of Denistry, from which he was graduated in 1890, having spent two years in outside work between his freshman and sophomore years.

After his graduation, he became demonstrator of operative dentistry and instructor in anatomy at his alma mater, at the same time attending night classes in architectural and mechanical drawing. Later in life, he studied metallurgy, drawing, chemistry and ancient and modern languages.

Dr. Prothero began practice in a southern state but remained there only a short time. In 1892, he started practice and began laboratory work in Chicago. In 1893, he was appointed superintendent of clinics in Northwestern University Dental School; in 1895, he became professor of operative dentistry in the American College of Dental Surgery, and, in, 1898, he was made professor of prosthetic dentistry and metallurgy in Northwestern University, after the two schools had merged in 1896. In 1918, he resigned and re-entered private parctice. For the past ten years, he had specialized in radiology, and at the time of his death, he was professor of that specialty at Northwestern University and professor emeritus of prosthetic dentistry. His chief contribution to dental literature was a volume on prosthetic dentistry, and he was in great demand as a lecturer and demonstrator before the various societies.

Dr. Prothero was a member of the Chicago Dental Society, the Illinois State Dental Society and the American Dental Association. He was also a member of Delta Sigma Delta and Omicron Kappa Upsilon and a Fellow of the American College of Dentists. He was a past president of the American Society of Radiographers.

His widow, Dr. Kate D. Prothero, survives him.

BUREAU OF CHEMISTRY

ANACIN

Imposing on the Public Through Unthinking Dentists

ANACIN, one is told, is a "combination of drugs which are antirheumatic, antipyretic and analgesic." It is marketed by the Anacin Company of Chicago. The vendors of Anacin use an old method of marketing their product by supplying unthinking members of the profession with so-called professional samples for "clinical use" among their patients. These unthinking members thus act as the tools of the Anacin people in giving to the product a certain mysterious glamor of professional authoritativeness, and are thus responsible for aiding in its uncritical and indiscriminate use by the public in medical as well as dental conditions. Yet on the other hand, lest the public be deprived of Anacin—and the company of subsequent profit—the product is also advertised extensively to the public.

The claim is made that:

> Anacin—the scientifically balanced formula of proven, non-narcotic remedies compounded to give prompt, safe, sustained relief without affecting the heart or disturbing the stomach.
>
> Anacin also relieves headaches, toothache, earache, neuritis, colds, influenza, neuralgia, rheumatism.

Anacin has a history. It was formerly marketed from Minneapolis by a concern that dealt in dental supplies. It has been reported that about January, 1927, the trademark was transferred to the Van Ess Laboratories of Chicago. The Van Ess Laboratories, it will be recalled, were responsible for the exploitation of "Van Ess Special Dandruff Massage" and "Van Ess Liquid Scalp Massage." Concerning the Van Ess products, *The Journal of the American Medical Association,* in 1923, stated in part:

> Even after the Van Ess concern has paid its advertising bills, and they must be heavy, there should still be a handsome profit at selling twenty cents' worth of kerosene and alcohol for forty-eight dollars.

The Anacin Company is stated to be a subsidiary of the Van Ess Company.

The propaganda concerning Anacin has not been free from misinformation for some time. According to *Hygeia* of April, 1928, one learns:

> Anacin originally was put on the market by a manufacturer of dental supplies that had as its president a dentist. A year or two ago this man declared that Anacin was the formula of a reputable Minneapolis physician (whose name he gave) and had been worked out by this physician and "his associates at the University of Minnesota Medical Department." Later the same man admitted that this statement was false and then declared that the Anacin formula was said to have been suggested by a number of prescriptions compounded by the Minneapolis physician previously referred to.

With the background for the promoters disposed of, the claims for the product can now be discussed. Claims made for Anacin are:

Anacin is a combination of synergestic drugs, scientifically compounded so that the well-known pharmaceutical principles of these drugs are available to relieve pain and reduce fever in certain conditions without any fear of depressing the heart or upsetting the stomach.

Evidence of the pure character of the drugs employed in the manufacture of Anacin is shown by the rapid way an Anacin tablet will dissolve in water.

Anacin contains para-acetphenetidin, acetylsalicylic acid, caffeine alkaloid and quinine sulphate. Each drug has a particular action of its own, and combined, they have a synergestic action, which insures the maximum of relief without any untoward or ill after effects or reaction.

Acetphenetidin (introduced as phenacetin) and acetylsalicylic acid (introduced as aspirin) are well-known drugs. So, too, is quinin and the drug caffein, chief constituent of coffee and tea. Such a mixture, therefore, on the face of it, has nothing unusual about it to command overweening respect. In spite of the claimed "synergestic action" for Anacin, no evidence beyond the claims made by the vendors of the product has appeared, according to our records. And why should it? It is simply a mixture of well-known drugs, irrationally compounded and representative of the days when therapeutic prescribing was not so scientific as it should have been.

Thinking members of the profession will ask themselves whether the claimed therapeutic actions are not obtainable by any of the drugs alone; all of which can be obtained under pharmacopeal names and definite standards of purity. The use of such a mixture as this retards scientific therapy in dentistry. Indeed, the Council on Pharmacy and Chemistry of the American Medical Association has expressed itself in regard to unnecessarily complex mixtures in this wise:

The Council holds that complex mixtures of remedial agents are from every point of view inimical to therapeutic progress and therefore to the public welfare.

They are especially objectionable because it is impossible accurately to determine the effects which follow the simultaneous administration of a number of drugs having dissimilar actions, and because the practice of prescribing such mixtures tends to discourage careful consideration of the special needs of individual patients without which there can be no rational drug therapy. On the contrary, with the use of such mixtures therapeutic treatment becomes haphazard and mere guess work.

Therapeutic authorities point out that drugs such as acetphenetidin, acetanilid, acetylsalicylic acid, quinin and caffein are active drugs and their doses should be regulated with particular reference to the tolerance and the dosimetric requirements of the patient. As the Council on Pharmacy and Chemistry of the American Medical Association has emphasized, "It is irrational to administer in fixed proportions such drugs as caffeine and either acetanilid or acetphenetidin, because their rates of elimination are not all the same, and their initial doses vary with different patients."

The indiscriminate distribution of such mixtures on the part of the profession is apt to be a boomerang. The claim is made that no untoward or ill effect or reaction follows the use of Anacin, a component of which is acetphenetidin. It would be interesting to learn how the Anacin Company rendered phenetiden harmless. This claim is not in harmony with the findings of the United States Department of Agriculture Bureau of Chemistry, which, in 1909, found that of 1,169 cases of poisoning by harmful drugs (acetanilid, antipyrin and acetphenetidin), 165, or more than 14 per cent, were due to acetphenetidin (a component of Anacin). Of 54 cases of death, 10, or more than 18 per cent, were due to acetphenetidin. Of 169 cases of habitual use of these harmful drugs, 18, or more

than 10 per cent, were attributed to acetphenetidin. Thus, the dentist who hands out Anacin (so liberally supplied by the vendors) makes himself a party to the further use of these drugs by the public; for it is easy to imagine that, subsequent to a visit to the dentist, the patient will recall the name on the envelop and, acting on presumably professional endorsement, resort to self-medication for different "aches" without realizing the dangers inherent in the use of such mixtures when not under the immediate observation of the physician or dentist. This may explain the motif of the Anacin people in so liberally supplying the profession with samples. Equally bad is the fact that there is no justification for the use of such a mixture. If the dentist desires to prescribe an analgesic, it is in his purview to prescribe scientifically that which is indicated, but to prescribe an unscientific mixture under a coined, meaningless name is not conducive to therapeutic progress.

As the complete qualitative and quantitative formula of Anacin does not appear on the trade packages, the product was submitted to the A. D. A. Chemist for examination.

A. D. A. CHEMIST'S REPORT

"One (1) original package of Anacin (The Anacin Company, Chicago, Ill.) was purchased on the open market for examination. The following appeared on the label:

Anacin
Non-Depressent
Non-Narcotic
Anodyne Compound

Each tablet contains:

Para-acetphenetidin (acetanilid derivative) 3 Gr. per tablet, Quinine Sulphate, Acetylsalicylic acid, Caffein.

Adult Dose

For the relief of pain in such conditions as Headache, Toothache, Neuralgia, etc. One or two tablets with water, followed by one tablet per hour until relieved.

"The dosages of quinin sulphate, acetylsalicylic acid and caffein are not stated, although these all are active drugs.

"The package contained 100 white tablets, the average weight of which was 0.45 gm. (approximately 7.0 grains), with a variation of 8.8 per cent below to 6.6 per cent above the average weight. When the tablets were crushed and powdered, an odor resembling acetic acid was observed, although the statement is made that 'impure specimens of so-called acetylsalicylic acid may contain free acetic and salicylic acids as may be observed from the pronounced acetic odor.' Further, an application of the U. S. P. X. Test for salicylic acid as an impurity in acetylsalicylic acid would indicate that the amount of free salicylic acid as an impurity was excessive.

"Qualitative tests indicated the presence of acetylsalicylic acid, quinin, caffein, acetphenetidin and sulphate.

"Quantitative determinations yielded the following:

Acetylsalicylic acid	42.0 per cent
Quinin sulphate	3.2 per cent
Caffein	4.5 per cent
Acetphenetidin	47.7 per cent
Moisture and undetermined (by difference)	2.6 per cent

"On the basis of the foregoing, each tablet would contain essentially:

Acetylsalicylic acid (aspirin)	0.19 gm. (3.0 grains)
Quinin sulphate	0.015 gm. (¼ grain)
Caffein	0.020 gm. (⅓ grain)
Acetphenetidin	0.217 gm. (3.3 grains)

Here, then, we have the spectacle of even some reputable members of the dental profession inadvertently aiding a "patent medicine" concern to use the pro-

fession as a screen for the advertisement of its product and thus unconsciously being parties to the indiscriminate sale to, and the uncritical use by, the public.

Dental practitioners who make themselves parties to the use of such irrational and unscientific mixtures as Anacin, parading under meaningless and noninforming names, hinder the development of a scientific materia dentica. If the profession is not to be remiss in its duty to the public, it must take the means and action to discourage the individual members of this responsible profession from being "used" as the means of exploiting such "patent medicines," with the inherent consequent dangers to public welfare. Reprehensible is the nefarious method of exploitation wherein the profession is imposed on by the commercially minded, and made peddlers — without charge—of an uncritical mixture essentially of aspirin and phenacetin: three grains of acetylsalicylic acid, 3 grains of acetphenetidin, and such small amounts of quinin (one-quarter grain) and caffein (one-third grain) as to be ridiculous from the standpoint of dental therapeutics.

Your Children
May be relieved from Colds, Fever, Headache, and the agonizing pains of Toothache and Earache.
Average Child's Dosage of An-a-cin may be safely administered to otherwise normal children for prompt relief of these conditions.
AN-A-CIN — the clinically proven balanced formula of proven non-narcotic remedies that does not affect the heart.
For Children and Adults of All Ages
Non-Narcotic
Non-depressant
This Handy Box 25¢
AN·A·CIN
Stops Pain!

Fig. 1.—Copy of an advertisement which appeared in a large metropolitan newspaper.

Sad to state, such "patent medicines" as Anacin have been allowed the privilege of exhibiting at conventions of dental societies. Thus, organized dentistry too has been involved by the "patent medicine" promoter. But it is hoped that with the awakening of the profession to the requirements of a scientific materia dentica, these nostrums will not be allowed to be clothed with a dignity which they by no means deserve. Encouraging is the fact that the leaders of dentistry are revolting against such exploitation.

Don't forget to Register for FREE Anacin
Dispensing Service for 1928
Dear Doctor
We want you to receive Anacin regularly for your own use. No strings attached to this offer. Just sign up at the convention—Booth No. 114—or write us.
Anacin is a great boon dentists tell us. It makes work easier for the patient and the doctor.
What It Is—What It Does
Anacin is a non-habit forming compound for the quick relief of pain. It does not disturb heart or stomach. Quick and safe, it can be used for children and invalids.
Dentists find it an invaluable aid and over 60 per cent are now hearty Anacin Boosters. We would like to make you another friend, Doctor.
THE ANACIN COMPANY
30 E. Kinzie St., Chicago, Ill

Fig. 2.—An indirect but cheap and effective way of reaching the public.

AFTERMATH

After this article was in type, Notice of Judgment (No. 15819—issued April, 1929) was issued by the U. S. Department of Agriculture, charging misbrand-

ing of Anacin in violation of the Food and Drugs Act, and declaring the claims for Anacin *false* and *fraudulent*.

The government alleged that Anacin was misbranded in that the package containing Anacin failed to bear a statement on the label of the quantity or proportion of acetphenetidin, a derivative of acetanilid, in such a way as to comply with the purpose intended by the Food and Drugs Act.

Misbranding was alleged for the further reason that the following statements regarding the curative and therapeutic effects of Anacin "were false and fraudulent, since it contained no ingredient or combination of ingredients capable of producing the effects claimed:

"Usual adult dose for headache, toothache, earache, neuritis, neuralgia, colds, rheumatism."

"Without any fear of depressing the heart . . . It has no depressing action on the heart, and can, therefore, be safely administered to children and invalids . . . without an untoward or ill after effect or reaction."

One wonders what excuses periodicals which continue to carry advertisements of Anacin will offer in the light of the government's action.

BUREAU OF DENTAL HEALTH EDUCATION

INTERNATIONAL DENTAL CREDENTIALS

By ALPHONSO IRWIN, D.D.S., Camden, N. J.

WHEN a dentist desires to locate in a foreign land, certain official formalities must be observed, of a local, a national and an international character. The dentist may deem the legal regulations of his own country burdensome, but he will find that these burdens are multiplied abroad, rather than diminished, as some seem to imagine they may be in a foreign country. This increase in complications should be distinctly understood at the outset, weighed impartially before arriving at a decision, and met intelligently, if a dentist decides to locate in another land.

It should also be clearly understood in the beginning that these statements do not apply to all foreign countries, but are true of the most desirable ones from a professional point of view. The civil regulations for dental licensure are usually strict in proportion to the culture, refinement and wealth of the nation involved. The higher the grade of intelligence reached by a progressive civilization, the more exacting becomes the demand for the greater proficiency of professional men, by law and practice, and on the part of officials representing such a civilization.

The credentials referred to generally as being necessary to a foreign-born dentist, an American, for instance, prior to entering on the practice of any profession (but dentistry more especially) in any foreign country, usually consist of the following:

DIPLOMAS

A diploma from a reputable dental college, recognized as such by the foreign country, must be signed by the professors of the said college and countersigned by the provost, dean, secretary, registrar or rector of the school. A state university diploma is always preferred, and is usually the only one accepted.

LICENSES

A license, properly signed, from the board of dental examiners of the state or country wherein the dental college is located must be presented. This state license is indispensable and validates the dental diploma abroad. Both diploma and license should be certified to by either the secretary of state or governor of the state in which the applicant resides.

PHOTOGRAPHS

Recent unmounted cabinet-size photographs of the candidate, endorsed by the dean or registrar of the college, after being attested to by a notary public as being a facsimile of the rightful possessor of the foregoing diploma, are always required. Many state boards in the United States require two photographs, and some civil officials require two photographs also. Passports must be accompanied by

three photographs, and they are absolutely essential to an alien entering a new country. It should be observed that state officials are subject to change, and therefore departments rather than individuals should be addressed. Intercourse with government representative should be coached in the official language of the foreign country concerned, especially in written communications.

VISES

The American applicant for a foreign license in dentistry must obtain from the Secretary of State at Washington, D. C., his visés to the foregoing credentials and attach the official seals of state thereto. The consul-general located in New York City, representing the foreign nation concerned, should be satisfied as to the authenticity of the credentials, the identity of the applicant, his right to the possession of all credentials produced, and his signature attached to said credentials, certifying to the correctness of the facts stated.

On his arrival in a foreign land, an alien dentist must apply to the secretary of state at the capital city. The secretary of state of the country in which the foreign-born dentist expects to locate, after the credentials have been translated into the language of the country by an official interpreter, is solicited to endorse the document after the nearest consul resident in the foreign country representing the native land of the applicant certifies to the genuineness of the document and the authenticity of the signatures attached thereto. The secretary of public instruction is often substituted for the secretary of state in the foreign land; or, as in Japan, the minister of home affairs, or it may be the minister of foreign affairs who will attend to these formalities according to the provisions of the law, in the country involved.

WITNESSES

Two witnesses, residents of the foreign land, preferably freeholders, who are not minors or related to the candidate are required to identify the applicant and vouch for him as the rightful possessor of the credentials.

STANDARDS

Credental educational qualifications of a certain standard are required by some foreign countries, which designate such requirements specifically, in their instructions. These may include a graded systematic course of specified instruction covering periods ranging from eight to twelve or sixteen years according to the established educational standards of the country. The training of a professional often begins at 9 years of age.

EXAMINATIONS

After all these official preliminaries have been observed and carried out, in many foreign countries the applicant is required to take at least one year's course in the foreign dental school nearest the place where he desires to locate, and pass the final examinations, paying all the fees required, in the same sums the native student pays for the course, before he is granted a license. If, on examination, he is not found qualified to enter the senior year, he must take a two years' course or the entire course according to his rating, based upon such examination.

LANGUAGE

These steps presuppose a practical knowledge of the official language of the country, without which the candidate cannot hope to achieve success, either in obtaining a license or acquiring a practice, for serious and even fatal blunders may occur in treatment of disease through misunderstandings from ignorance of the language. Furthermore, it should be borne in mind that the grant-

ing of licenses to practice dentistry abroad, while dentists may conduct the technical examination, is under medical supervision by a mixed board composed of physicians and dental surgeons; also, the universities, medical and dental schools are under governmental control, receiving appropriations which help to defray the expense of conducting them. In other words, they are state institutions, with the supreme authority vested in the head of the government, whose decisions may be appealed to above the decisions of the medical council.

While we do not represent that each and every one of these formalities topically specified must be observed by every foreign-born dentist applying for a license to practice dentistry, in each and all foreign lands under all circumstances, we do assert that any alien dentist who wishes to establish himself firmly and legally in a foreign country should omit only such procedure as he is advised to omit, with the consent and approval of the proper officials of both countries. Furthermore, he should scrupulously observe all instructions imparted to him by the dignitaries of the governments represented, the faculty of the dental school in which he may become a student, or with whom he may become associated, and the examiners who shall conduct the final test of qualifications for practice in that particular country. For the alien dentist to secure "release" from one or more of these requirements is rare, as is also "securing an 'approbation' from a state government."

NATURALIZATION

Many foreign countries require the alien dentist to become naturalized, take the oath of allegiance and become a bona fide citizen of the land where he takes up his domicile for the purpose of practicing his vocation. In some foreign countries, this requirement is insistent and must be complied with. A few foreign countries will not grant a license to any dentist unless he is native born and educated in the institutions of his country. Such a prohibition is reported in Portugal, Spain and their protectorates or colonies, in the Ottoman republics, and with reservations in several other lands, such as Denmark, Holland, Sweden, Norway and Switzerland. In these last named countries, the reservations are so qualified as to make the licensing of an alien dentist almost impossible. The predental educational requirements alone bar the alien dentist from legal practice, with possibly an exception in case he wishes merely to become an assistant licensed to a native dentist.

The foreign-born dentist who is provided with all of the foregoing credentials is well equipped and, his other qualifications being equal, should achieve gratifying success, particularly if he is so fortunate as to have been trained in and graduated from a standard Class A dental college located in the United States of America. Such credentials are usually letters of introduction to professional, social and financial circles of the most desirable character, in foreign countries.

UNLICENSED ALIEN DENTISTS

Unlicensed alien dentists may be divided into four classes:

1. The dentist. In a few instances, an alien dentist, thoroughly qualified, has been permitted to practice dentistry in a foreign city in the colony speaking his own language, such as an American in Paris, Rome, Berlin, Vienna, Dresden, Stockholm, Leningrad, Tokio or some other city, possibly in the Orient, in Africa or in some island of the seas. The number who pursue their vocation under these circumstances is limited. Some of them possess an international reputation.

It might also be added that the time is passing for making this kind of venture abroad, pending the establishment of a new order of things under the reconstruction era, now under way in the most desirable countries abroad. "Dispensations" are granted only by an emperor, a king, a president or some legally designated authority (as in Norway, Sweden and Great Britain).

2. The assistant dentist. The graduate may become an assistant dentist in the office of a native licensed practitioner, on contract, with a view to complying ultimately with the license regulations enforced therein, and entering into partnership with his former employer or succeeding him in the practice, on specific and prearranged terms. Unlike the United States, which stipulates that the assistant dentist must be licensed, foreign countries permit the employment of alien qualified assistants who are unlicensed in their adopted country, but possess a license in their native land.

3. The demonstrator. The graduate dentist who enters into an agreement with a foreign dental depot or firm to perform specific work for a certain length of time, for a fixed salary or on a commission basis, or for a salary and commission may or may not enter on the general practice of dentistry. These contracts must be drawn up correctly according to the laws of the foreign country concerned beforehand or misfortune may overtake the contracting party.

4. The itinerant dentist. The independent unlicensed foreign-born graduate, or nongraduate, may take a chance and go into practice for himself in the most promising location. Isolated cases are cited wherein such practitioners have flourished, for a time at least; but we also know of instances in which the unlicensed dentist has been compelled to relinquish a lucrative practice and make a hasty exit, with financial loss. It should also be considered that the one who has achieved signal success is freely quoted; whereas, the larger proportion, who fail, are unheralded. "The American Dental Parlors" flourish for a time in some central or south American cities, but like the Arabs eventually "fold their tents" and "silently steal away."

PASSPORTS

Passports for alien dentists are indispensable, not only to pass the customs, but also in order to secure a license to practice dentistry in a foreign country from the vested department or authority.

COSMOPOLITAN TRAINING

We have met dentists from abroad who have received their predental and initial dental training in their native country (usually in European schools, though some have graduated in Japan and elsewhere) and have then come to the United States and graduated, after due course of instruction, from the dental department of a state university, lastly taking a course in Edinburgh, Scotland, or a London university, though occasionally elsewhere. The L.D.S. certificate granted by a recognized university is not now registerable in all parts of the British Empire, but is recognized and accepted for registration in many parts on compliance with all the local requirements; while the medical degree is desirable in all parts of the world, for permission to administer anesthetics, as well as for other reasons. In many foreign countries, a medical graduate must be present when a general anesthetic is administered, and, in some lands, when a local anesthetic is administered. This is a regulation which the skilled American dentist would scornfully resent. The temptation to acquire the conjoint

diploma of L.R.C.P. and M.R.C.S. and thereby enhance the value of their credentials is irresistible to some dentists of a studious disposition. Such training is of incalculable value in scientific and original research as well as expert legal work; to say nothing of its gaining the possessor the most fascinating and profitable clientele abroad. The men possessing such credentials have been highly developed and are fitted to fill any responsible position in the practice of dentistry in any quarter of the globe, by reason of their exceptional natural and acquired qualifications and cosmopolitan ideas. Such a training bestows a literary degree, two dental degrees and a medical degree. It presages a brilliant career for the man who has made a judicious use of his time and talents. In America, he will receive the practical education, acquire the technic, and become a well qualified dentist; while abroad he will be enabled to master the theory and absorb a broad culture, so helpful to any professional man, making him a master-mind in his chosen vocation, among the wealthy, influential or cultivated classes of society.

METHODS OF TRAINING

The French system of dental training, credentials and licensure commends itself particularly to the Latin races, and possesses some features which might be adopted with advantage by other nations.

The English method, when its graded exactions are adequately complied with, produces broadly educated professional men graduates well equipped theoretically to practice dentistry. The German standards have suffered an eclipse for obvious reasons. Their methods of dental training produce a practitioner ranging from the slick commercial German dental mechanic to the highest type of cultured professional dentists; while they make some exceedingly fine distinctions between the citizens practicing at dentistry. The Japanese mode, which is ideal for the Orient, is commanding increasing respect in the Occident for its simplicity and thoroughness. Japanese dental mechanics in particular exhibit phenomenal skill. The American system has outstripped them in producing men of initiative, ingenuity, practical efficiency, technic and operative and prosthetic ability. Each system confers appropriate credentials, in accordance with the established laws of their respective countries, and the dentist should acquire such credentials as are acceptable in the country of his choice, for pursuing the practice of dentistry, unbiased by culture, race, creed, color or form of government.

REACTIONS

It is a fact not universally appreciated that international credentials enforced through legal license regulations are the agencies which keep the educational systems of a nation tuned up to concert pitch in the effort to promote "dental efficiency"; while reciprocity is designed to cut the red-tape of duplication of examinations, complexities of the law and the multiplication of useless obstructions to practice. The last named are sometimes swept away by a tidal wave, or the inexorable relation of demand to supply, while a reaction is inevitable when the enforcement of the law becomes tyrannical. This reaction assumes many phases, not the least important of which is the advent and multiplication of the dental adviser, the so-called dental parlors, down to the blatant fakir. The credentials of such practitioners, whether of an international, national, state or local character, will require microscopic inspection, for they block the adoption of international reciprocity relations more than any other agent or agencies.

PHANTOM CREDENTIALS

In this connection, it should be remembered that the talent for manufacturing phantom credentials (we use the word "phantom" as a result of our own experience, as these credentials have a habit of mysteriously disappearing when most needed for close scrutiny) is not limited to perverted American genius alone. Other nationalities are also adepts in the art. We make this brief allusion to existing abuses in making application for a license to practice dentistry, because they justify the most punctilious regard for all the details which should be observed either in making out or accepting either national or international dental credentials. It should also stifle unfriendly criticism of dental regulations between nations which thoughtless men sometimes indulge in, to the detriment of their own cause and the destruction of international amity.

We have briefly outlined the maximum requirements of the most influential powers and hinted at the minimum possibilities open to the alien dentist trained in another country. They should be studiously examined before the final steps are taken toward acquiring a license to practice dentistry in a foreign location, if a dentist resolves to become successful in making the venture. The dentist should visit the foreign country of his choice beforehand and investigate the prospects thoroughly and minutely before making a final decision to locate there.

SORE SPOTS IN THE MOUTH*

By LE ROY M. S. MINER,† M.D., D.M.D., Boston, Mass.

THERE are a great variety of sores appearing in the mouth, and they are quite common. Many of them are simple in character, being important only because they may be of significance in indicating some physical disturbance. Occasionally, we find a sort spot which is of serious import.

One of the simplest spots appearing in the mouth is the so-called canker sore. This is usually extremely tender and may cause discomfort for a day or two, with enlargement of the glands underneath the jaw and in the neck. They heal up readily and are of no special importance. Frequent recurrences of these canker sores usually indicate a faulty diet, a disturbed digestion or some defective condition of the mouth itself. Those of you who suffer from these recurrent sores should stop eating candy and sweet desserts, and eat instead fresh fruits, unsweetened. Seek the advice of your dentist and your physician. Nature is trying hard to tell you that she is not being treated fairly.

The gum boil (abscess) is another condition that is commonly seen and usually causes no great trouble in the mouth itself. People are liable, therefore, to disregard this condition, but a gum boil means a chronic abscess in the bone which is discharging its pus into the mouth. The irritation produced by the pus around the opening is what we know as the gum boil. We all know that pus is a

*A radio talk broadcast during the Midwinter Clinic of the Chicago Dental Society, Jan. 16, 1929.

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poison and has possibilities of causing serious symptoms. In view of the possibility of harm to some other part of the body from this poison, gum boils should be attended to promptly. X-ray examinations to determine the location and extent of the infection should be made, so that the treatment can be intelligently given.

Vincent's infection, or trench mouth as it is popularly called, is another condition of importance, associated with multiple sores. It is important, first, because it is highly contagious, and, secondly, because it is increasing in frequency at an alarming rate in this country. If this condition is not thoroughly eliminated, permanent damage is done to the mouth and teeth. The disease occurs in both acute and chronic forms. As I have said, it is highly contagious. It is spread through kissing and through the use of common drinking cups, table ware, pipes, towels, etc. During the period of treatment, it is necessary to eliminate everything that is likely to produce irritation. Smoking should be stopped and extremely hot food and drink should not be taken. Expert dental attention is necessary.

Leukoplakia (which means white spots), or smoker's spots, are seen quite commonly in heavy smokers. They are not in themselves serious, but they indicate that an irritation is occurring and that Nature is attempting to protect herself against that irritation. Occasionally, these spots break down, as we say, and become malignant. Because of this tendency, leukoplakia has been called a precancerous lesion. If these spots appear, it is wise to reduce smoking or stop it altogether, and to avoid taking hot and highly seasoned foods. Sometimes, it is wise to have these spots removed. They are not, as a rule, painful. The lesions of syphilis are sometimes confused with leukoplakia. These two diseases are totally different in importance and significance, and the treatment also is therefore quite different, and consequently an accurate diagnosis is a prime requisite.

The most important sore spot in the mouth that we meet, from every point of view, is cancer, a malignant tumor. Because of its serious importance, I want to devote the rest of the time allotted to me to this subject.

Cancer of the mouth, or oral cancer as it is technically called, includes invasion by this disease of the tissues of the cheeks, lips, palate, gums and tongue. It is not a particularly entertaining or cheerful thing to talk about, but every cloud has its silver lining, and without discounting in the least its serious importance, it is fair to say that even this gloomy subject has its bright side. Many people, I find, have a hidden fear of developing cancer of the mouth. They frequently worry when there is no reason for it at all. I hope to bring a message of comfort to such individuals this afternoon. But the ravages of this disease are so appalling that I wish also to arouse to action all those who have developed sores in the mouth which show no tendency to heal with reasonable promptness. Neglect is an evil genius which puts its fatal touch on this disease.

Prevention of disease is the goal toward which all health agencies are striving. All efforts toward the prevention of cancer should receive the hearty support of everyone. Cancer week has for its purpose the spreading of accurate information, so that the people of the nation may become acquainted with the facts.

In cancer of the mouth, we have an advantage over cases located in the deeper parts of the body, because, from its earliest inception, the individual is conscious of the presence of a sore in mouth. It

may not be painful. Usually it is not in the early stages. It may be regarded as only a canker spot, or a cold sore, or some other simple condition. Perhaps the individual may feel that he has bitten his cheek or that he is smoking too much, but no matter what he thinks about it, he knows something is wrong and he knows this at a time when, if it is cancer, it is in a stage that may be successfully treated. Another advantage of a suspected cancer of the mouth which is closely related to the one already mentioned is that an actual diagnosis can frequently be made in this very early stage before the disease has had a chance to develop to a hopeless stage. Early diagnosis and early thorough removal of the growth constitute bright spots in this matter of cancer. In the case of a sore which cannot be positively diagnosed before operation, rather than lose valuable time by inaction it is better to consider the lesion guilty until proved innocent.

The two most important characteristics of cancer of the mouth are, first, its failure to show any tendency to heal even with help, and second, the persistent spreading of its growth. A well-known surgeon in this country, whose experience has been wide, has said that a cancer of the mouth growing for more than six weeks becomes hopeless. Time, then, is an important element in the favorable treatment of a case of cancer. When one has developed a sore in the mouth and has consulted a dentist or a physician about it and treatment has resulted in a reasonably prompt healing, there is no occasion to worry about cancer. Pain, even of a persistent character, does not necessarily suggest the presence of cancer, especially when no sore exists. This may help to relieve the minds of some of my radio audience. But when a sore has been treated for a week or two and various treatments have been given without success, and no satisfactory explanation for this failure can be made, it is important to remember, first, the characteristic persistency in the growth of cancer; secondly, the fact that local applications are of no avail; and, thirdly, the additional fact that the time margin of safety is very low. Therefore, the wise man or woman who has had a sore in the mouth, whether it has been treated or not, which does not heal within two weeks, will not fail to have it examined at once by some one familiar with this disease, for the purpose of establishing a diagnosis. If this is done and the sore proves to be a cancer, thorough treatment promptly given offers a good chance to completely eliminate the disease.

"What is the treatment?" I hear some of you asking. Surgery, radium and the x-ray are the three accepted measures of the present day. Carefully kept records show that early operation will eliminate the disease in an encouraging percentage of cases. Surgery followed by radium or x-ray is usually more effective than surgery alone.

The specific cause of cancer has not yet been discovered, but large sums are being spent all over the world for researches in this field, and all these efforts should be liberally supported. The discovery of the cause may become a fact at any time. Meanwhile, we know that irritation seems to be of considerable importance as a causative factor. A normal, healthy mouth, free of irritation from within and without, is relatively immune to cancer. The prevention of all irritation in the mouth, such as might come from sharp roots or fillings or from mechanical inserts of any kind is important.

Irritation from without, such as might come from too much smoking or taking foods too hot, should also be guarded against. Moderation is a most desirable virtue: moderation in eating, in smoking and in all other phases of living may go far toward reducing the frequency of cancer.

HOW THE TEETH AND JAWS GROW*

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I HAVE been asked to tell you something about how the teeth and jaws grow. The subject will not be presented in a technical way, but I shall try to tell you what should be done to induce these tissues to grow and develop in such a way as to be strong, durable, good looking and capable for a long period of efficient use.

It is logical to start at the beginning: You probably have but little idea as to how early in the cycle of life these tissues are started in the formative process, and therefore how early thought should be given to the factors that govern the destiny of the "gate-way to the human body."

Long before a child is born—when the little body is but five weeks along life's cycle—the teeth have started to form, and it may surprise you to learn that this development is taking place considerably in advance of the formation of the bony structures that are to support the teeth. The tooth is laid down first and then the other highly specialized cells change the cartilaginous matter to a more or less bony matrix, which will eventually become the true bony frame work as we understand it to be.

At birth, the crowns of the twenty deciduous or temporary teeth are all quite well formed, and the enamel caps of the permanent teeth, except the third molars, or wisdom teeth as they are commonly called, are started. The jaw bones gradually form around the teeth, and each tooth becomes loosely enclosed in a cavity or crypt. The tooth root is growing in length, the crypt fills in with bone, and meantime a third interesting and important tissue is developing, the peridental membrane. This membrane surrounds the root and furnishes a portion of the nutrition to the grown tooth. It also acts as a cushion while we are chewing, a shock absorber so to speak, to relieve stress in biting hard substances. In passing, it may be well to mention that it is the "breaking down" of this membrane later in life that is known as pyorrhea.

As the teeth develop and approach the surface, there is a decided thickening and blanching of the gum, frequently with considerable discomfort, which causes the fretfulness quite common in a baby's teething period. Most mothers have observed that the baby likes to bite on something hard at this time; hence, the "teething ring" to help the teeth through the gum. It is sometimes necessary to have the gum lanced by the dentist to relieve the imprisoned tooth.

The lower incisors are usually the first teeth to come through the gum, erupting,

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as we say, at about 6 months of age. For the next twenty or more months, the jaw bones are the scene of great activity; for by the time a child is from 2½ to 3 years of age, all twenty of the baby teeth should be in place, ten above and ten below.

The lower teeth usually erupt in advance of their mates in the upper jaw, and for this reason dentists speak of the lower jaw as the pattern over which the upper is formed—its size and shape influence the size and shape of the upper jaw; and it is well to remember that if the lower jaw, from any cause whatsoever, in either the temporary or the permanent teeth, is deformed or underdeveloped, the upper jaw will be proportionately underdeveloped.

At birth, the lower jaw bone forms a nearly straight line from its joint or hinge to the tip of the chin, with the lower part of the face decidedly short and undeveloped as compared to the upper part of the face and head. As the teeth erupt, the jaw changes its form at the angle, and the whole lower third of the face develops proportionately. With the loss of the teeth in advanced age, there is again a shortening of the lower face with a loss of bone structure resulting in jaws quite like those of infancy.

There is a surprising idea that the crowns grow larger after eruption. This is incorrect; for by the time the crown portion is through the gum, the only further growth is in the length of the root. It is this growth that causes absorption of the gums over the crown and allows the tooth to erupt.

Anxious parents often ask about the points or "saw edges" on a child's front teeth. If these little points are smoothly rounded cones, they need cause no worry, for they are the natural marks or remains of the several parts or lobes that have united to form the crown, and in a short time will wear away as the teeth are put to use. This condition must not be confused with the decided roughness and pitting of the enamel which have been caused by early malnutrition or disease. The eruptive fevers of childhood are liable to affect the teeth thus, and improvement in appearance can be had only through skilful dentistry at the proper time.

In this problem of growing sound teeth and sturdy bone to support them, it is necessary to stress the fact that much depends on the health and right living of the parents. The responsibility of the mother is particularly great because the child obtains its nourishment from her during a period of very rapid growth. Her foods and surroundings should be well planned, for they will have much to do with the character of bone and teeth and the general health imparted to the infant. If the mother's diet does not supply sufficient mineral matter, these minerals will be drawn from the tissues of her body, in which case the bones and teeth of both mother and babe are liable to suffer.

There is no food for an infant, when all conditions are normal, that is equal to mother's milk, and there is no method for the child to receive this food that can be compared to breast feeding. The development of the jaws, teeth and face due to this particular muscular action is unequaled. The quality of food, the exercise and the stimulation to growth cannot be had in artificial feeding.

When solid foods are introduced into the child's diet, those should be selected which, along with proper nutritional elements, will require thorough mastication. Bone is a more or less plastic material and its shape or form is largely influenced by the stimulation which comes from

exercising the muscles. Therefore, the more energetically the food is masticated and the muscles worked, the better will be the development of the dental arches and the bones of the face.

Between the fifth and sixth year, a second great change in the dental apparatus starts. The first permanent molar erupts directly behind the baby molars. This is perhaps the most important tooth of all and incidentally the most neglected because so many people do not realize that it is the first member of the permanent denture to appear. With its eruption, the jaws are lengthened and the height of the lower third of the face is increased.

About the same time (at 5 years), if development is taking place normally, another great change is starting. A decided spacing should be taking place between all of the front teeth both upper and lower. This spacing indicates a lateral growth or widening of the jaws and face and is Nature's provision to enlarge the dental arches in order to allow the larger permanent incisor teeth to erupt in regular alinement. If these developmental spaces are not appearing at from 5 to 6 years of age, you had better consult a dental specialist who straightens teeth.

The loss or shedding of the baby teeth at from 5 years of age to about 10 o[illegible] is a physiologic process but one which does not always take place in accord with the usual expectations or ages. If your child is in the hands of a competent dentist, the latter can inform you as to whether the shedding of the teeth is progressing properly or not. It is just as important that baby teeth be lost or removed at the proper time as it is for them to be retained to their full term. If they are lost too soon, development is retarded by the muscles of the lips and cheeks compressing the still soft bones and settling the arches together; and if they are retained too long, they may direct the permanent successors to a wrong position. These are conditions that call for a nicety in judgment not always displayed.

The second permanent molar takes its place in the dental arch about the twelfth year, and the third molar, about the eighteenth year. These teeth are further increasing the size of the jaws and face.

The remarkable change in facial balance between 6 years of age and youth, because of the growth of the teeth and supporting bone, can only be touched on here. These changes are intensely interesting and important, for so much in life depends on the face as an index to character and personality.

There are a number of conditions that tend to deform the bones of the mouth and produce irregular and unsightly teeth in addition to those which have been suggested here. Among the common ones are habits such as mouth-breathing, thumb-sucking, the use of pacifiers and nail biting.

Happily, these deformities can be corrected by skilful dentists, but prevention [illegible]ways better than cure. Someone has said "The health of the people is the supreme law." Within this idea, we find the foundation of all material, intellectual and spiritual success; for, without health, no people will ever attain greatness.

CURRENT LITERATURE

EXTRACTS AND ABSTRACTS

Occupational Poisoning in Manufacture of Luminous Watch Dials: In two special articles (*J. A. M. A.*, Feb. 16 and 23, 1929) H. S. Martland gives a general review of the hazard caused by ingestion of luminous paint, with especial reference to the New Jersey cases, first reported by Castle, Drinker and Drinker (*J. Indus. Hyg.*, August, 1925; abstr. *J. A. D. A.*, October, 1925). In discussing the jaw necroses, Martland says: "What, then, is the explanation of the extensive necroses in the mandible and often the maxilla seen in the early fatal cases, and absent, recovered from, or never a prominent symptom in the patients now alive? In some of these cases, this necrosis would precede the appearance of a severe anemia; in other cases, it would develop after the anemia had existed for some time. There does not seem to be any connection between the two. In our early work, in 1925, we were at a loss to explain the localization of this extensive necrosis in these bones. As the jaw necrosis was the outstanding feature of the early cases, our attention was fixed mainly on this symptom and little was paid to changes in the other bones of the skeleton, although an extensive coxa vara was noted in one case. The necrosis of the jaw was at that time thought to be due to local irritative radiation at the portal of entry, produced by clinging particles of radioactive substances on the gums, in the crevices and interstices between the teeth and the bone, activated by increased bacterial virulence of the organisms in the mouth caused by the irritative or stimulative effects of small amounts of radiation. These bones were thought to contain a greater amount of radioactivity than did other bones of the skeleton. It was not until I began to see the later cases that I realized that this was not the correct interpretation of the lesion. In the later cases, localized pain had occurred in other bones and destructive lesions were demonstrated by the roentgenograms. These lesions were particularly noticeable in bones exposed to trauma, like the scaphoid in the feet (tight shoe), and those carrying considerable weight, such as the spine and the heads of the femurs. These lesions so far have never become infected. Autopsies had also shown that the jaw did not contain any more radioactivity than did other bones. In other words, the lesions were quite evenly distributed over the entire skeleton, although in each individual bone the distribution, while diffuse, was distinctly patchy and concentrated in areas. This distribution was demonstrated by photographic methods. Furthermore, histologic studies of bones, in which during life there had been localized pain and a roentgenographic appearance unlike that seen in any other disease, showed that the marrow of these bones was replaced by a gelatinous, myxomatous, acellular loose fibroblastic tissue similar to that described by Ewing in bones exposed to heavy external irradiation. From these observations, it was more logical to interpret the jaw necrosis as being primarily due to a radiation osteitis similar to that occurring in other bones. Owing to the proximity of the mouth, which is the dirtiest part of the body, and to the fact that the jaw bones have embedded in them the teeth, which act as foreign bodies, and because of the common occurrence of pyorrhea and root abscesses the jaws are easily subjected to a superadded bacterial infection, in these cases in bones already the seat of a previous disease. The slightest dental intervention may start an intractable necrosis of these bones. In the patients now living, the severe anemias and extensive jaw necroses which were the outstanding features of the early cases have not occurred up to the present time. The disease is now present in a milder form, undoubtedly as the result of the natural uninfluenceable decay of the mesothorium, which formed the largest percentage of the original deposits. There is a consequent diminution in the amount of irritative radiation. The main symptoms now are crippling bone lesions, the result of radiation osteitis. While

it appears that many of these girls will escape a fatal outcome, one cannot be too sure of this, as many show considerable radioactivity at the present time."

Epithelioma of Right Superior Maxilla: The case of a man, aged 52, successfully operated on for an epithelioma of the right superior maxilla, is reported by Willy Meyer (*Am. J. Surg.,* March, 1929). The patient had had a sore on the alveolar border of the right upper jaw which had caused some pain for the two months previous to admission. The teeth in this region had dropped out of themselves. Examination of the mouth revealed various areas of leucokeratosis, percursors of cancer, and at the site of the ulceration, a papillomatous growth which had turned into an epithelioma. The Wassermann reaction was negative and the patient gave no history of specific disease. The characteristic appearance of the lesion and its rapid onset and growth confirmed the diagnosis of epithelioma. Resection of the right superior maxilla was carried out under ether-olive oil colonic anesthesia, which the author has found ideal in these cases. The feature of the operation which led to its being reported here was, in addition to the satisfactory cosmetic result, the method employed, which was proposed by George H. Semkin, to whose service at the New York Skin and Cancer Hospital the patient had been admitted, of temporarily angulating the external carotid artery during the operation so as to save blood and also conserve full blood supply to the flap for healing purposes. In a discussion following the case report, the question was raised as to the advisability of securing a full return of circulation in these cases. Dr. Meyer's reply was as follows: "I agree that ligation of main arteries in operable cancers is a wise procedure. It starves the tumor and retards its growth. If a cancer is operable, however, and radically removable with proper cancer technique, and the tumor is removed in toto, then ligation of the artery is of no value. If a reconstruction operation has to be added, it is of the greatest value and benefit to have the full blood supply entering the operative field at the completion of the operation. This important point was demonstrated in the case presented. Ischemia was brought about by the temporary angulation of the external maxillary artery instead of permanent ligation."

Effect of High Cereal Diets on the Growth of Infants: The growth and development of 1,200 Japanese and 869 Filipino infants in Hawaii were observed during the first two years of life. The diet of both races is high in rice and lacks milk, and the infants are breast fed. Three hundred and seventy-nine Hawaiian infants were also examined for skeletal development. A diet abundant in cereal without milk is compatible with a high birth rate, moderate infant mortality and good growth of the offspring during lactation for the first five months of life, when the infants are dependent on breast milk alone, if the mother's diet includes suitable, adequate supplementary food other than milk. After six months, when the infants have reached the age when additional food is needed, the gain in weight is not so rapid as when cow's milk is used as a supplementary food in the diet of infants. Carpal centers develop early in the infants in Hawaii. The subtropical location of the Hawaiian Islands provides sufficient ultraviolet rays in the sunlight to give almost complete protection against rickets, even when the diet of lactating mothers and of infants under 2 years of age does not contain milk. Rickets is extremely rare and is mild when it occurs. The eruption of deciduous teeth occurs early in Japanese and in Filipino infants. It is earlier for the Japanese than for the Filipino infants. —V. B. Appleton, in *Am. J. Dis. Child.,* February, 1929.

Transmission of Ultraviolet Light by Different Types of Glass: It appears from the growth curves, the calcium and phosphorus content of the blood serum, the roentgenograms, the pathologic studies and the ash content of the bones that practically as much ultraviolet light passes through Corning glass as is obtained by direct irradiation. Vitaglass, on the other hand, permits the passage of only a certain fraction of the necessary ultraviolet rays. Ordinary window glass may be considered virtually useless in this respect, owing to the fact that the animals irradiated under this type of glass showed practically as marked a degree of rickets as those which did not receive any irradiation.—E. T. Wyman et al., in *Am. J. Dis. Child.,* March, 1929.

Cancer of the Mouth: I have become rather enthusiastic in the treatment of cancer about the mouth, since I have been depending almost entirely upon the surface application of highly filtered radium, applied both internally and externally during a period of about a month, on the principle of the "saturation

method." No definite rule of technique can be laid down, because no two cases are alike, but we treat to the limit of normal tissue toleration, using gamma rays filtered through the equivalent of from 2 to 4 millimeters of lead. I have not used this method long enough yet to give satisfactory statistics, but I get the impression that in the average case there should be a 50 per cent chance of complete recovery and in the early case there should be a 75 per cent chance. I have worked with cancer of the mouth for thirty years, but my results now are so much superior to all methods used by me previously that I feel like apologizing for my enthusiasm. —G. E. Pfahler, in *Illinois M. J.*, March, 1929.

Diagnosis of Mixed Salivary Gland Tumors: R. I. Hiller (*J. A. M. A.*, February 23, 1929) reports a case of mixed tumor of the left submaxillary gland, in a man, aged 38, which had been present for eighteen years, and which gave a definite response to the oral administration of potassium iodid, the tumor first increasing and later decreasing in size. The author suggests that this response to iodid therapy may prove of value in the differential diagnosis of tumors of aberrant salivary gland tissue.

Agranulocytic Angina Following Prophylactic Typhoid Vaccination: Bromberg and Murphy (*J. A. M. A.*, April 13, 1929) report a case of agranulocytic angina in a woman, aged 50, who had received a prophylactic typhoid vaccination a few weeks previously. The first injection was attended with malaise and fever, which developed within six hours and persisted for two days. A second injection eight days later was followed within four hours by malaise, fever and prostration. Four days later, the patient complained of sore throat and moderate gingivitis with swelling and tenderness of the glands below the jaw. Vincent's organisms were found in a direct smear. Leukocytes numbered 5,400. During the next five days, an ulceration with a necrotic base appeared around the left upper molars, spreading steadily with hemorrhagic margins. The uvula became intensely edematous. A small pustule at the right nasal orifice developed into a cellulitis which involved most of the nose. In addition, when the patient was admitted to the hospital, there was a necrotic ulcer in the hard palate, cervical adenitis, a precordial systolic murmur and an area of induration on the left labium majus. The course in the hospital was one of progressive decline, and the patient died four days after admission, of bronchopneumonia. All infected foci had spread rapidly, and periods of delirium were followed by stupor and coma. The authors conclude that since protein shock has been shown to be accompanied by blood changes analogous to those demonstrated in this case, the agranulocytosis may have resulted from an overwhelming foreign protein reaction in a sensitive individual.

Carcinoma of the Mouth: Radiotherapy has its most brilliant achievement in the results from the use of implants of radium emanation or radon in carcinoma of the mouth according to L. F. Weber and W. A. Pusey (*Illinois M. J.*, March, 1929). Both small lesions and extensive, apparently hopeless, lesions are best treated in this way. Should metastasis result, the treatment is warranted because of the comfort of a clean mouth to the patient and the relief from the torture of slow death from mouth carcinoma. Treatment by radium or radon should supplant surgery in these cases because results are better and the mutilation of surgery is avoided. Epithelioma of the lower lip alone of mouth conditions is better treated by other methods. Cancer of the tongue and the buccal membranes show best results when treated early and while the lesion is yet localized. Cheek lesions are fortunately early diagnosed and show favorable results. In some of these cases, radium or the roentgen rays are used to "cross-fire" from the outside. It is believed that clinical diagnosis is of more value in these cases than microscopic, as a negative microscopic test does not rule out maliganacy. "Inflammatory and epithelial growths of the mouth and skin are often difficult to differentiate from malignant lesions by means of the microscope." The insertion of the "seeds" containing radon "the highly radio-active emanation in the form of gas which is given off by radium" is a simple process and the seeds are commercially obtainable and inexpensive, from ten to fifteen being used in the most extensive cases. A large initial dose is given to destroy the growth before extension takes place. The tendency now is to treat the cervical nodes conservatively. Interference with the natural defense offered by the lymph glands must be cautious. When the nodes are not palpable, the front and sides of the neck are treated with the filtered roentgen rays. Glands that are malignant are best treated by surgical removal, roentgen-ray therapy being used

before and after operation. In the hopeless, inoperable cases, the rays are used as palliative treatment.

Irradiated Ergosterol in Rickets: Report of a study on the treatment of rickets by irradiated ergosterol is made by Wilkes, Follett and Marples (*Am. J. Dis. Child.*, March, 1929.) Twenty-four rachitic infants ranging in age from 9 weeks to 2 years were studied. To eliminate the favorable influence of the summer season, experiments were conducted from January to May, with the exception of two cases studied in June. No other antirachitic measures were employed. Blood serum calcium and phosphorus were determined weekly and roentgenograms of the extremities were obtained practically as frequently, with clinical histories. The study, with results, is summarized as follows: 1. Twenty-four infants with rickets, varying from mild to severe forms, were successfully treated with irradiated ergosterol. 2. Two infants with tetany were cured by irradiated ergosterol. 3. The irradiated ergosterol, in the form of a commercial preparation, was well taken and well tolerated. Two of the patients had severe bronchopneumonia, and several had previously refused or vomited cod-liver oil. 4. The commercial preparation used retained its potency for at least six months in one instance. 5. Reports of other authors who used irradiated ergosterol are confirmed. It was found that irradiated ergosterol requires about three weeks to cause serum blood phosphorus and calcium concentration to return to normal, about the same time for roentgen-ray signs of healing of the bones to appear, and about six weeks for complete healing. These averages compare favorably with those obtained with cod-liver oil, quartz light and egg. 6. Craniotabes was healed within an average of one month in the series of twelve patients. In individual cases a period of from twelve days to eight weeks was required. 7. Healing of the rachitic bone took place in one patient with bronchopneumonia, despite a low serum phosphorus and calcium product of 20.5 mg. per hundred cubic centimeters. 8. Caution may be necessary in using irradiated ergosterol in patients with tetany who show a high blood phosphorus content. 9. Irradiated ergosterol is not a perfect preventive for rickets. Craniotabes developed in one patient receiving prophylactic doses of irradiated ergosterol. This healed promptly when the ergosterol was continued in unchanged amounts.

Operation for Tic Douloureux: Partial section of the sensory root at the pons by the subsellar route was performed successfully by Dandy (*Arch. Surgery*, February, 1929; abstr. *J. A. M. A.*) in a considerable number of cases and was followed by relief of pain. The advantages of such partial section are as follows: 1. Immediate postoperative corneal disturbances are uniformly absent. 2. The motor root is always preserved. 3. Sensation, approaching the normal, is retained over the entire domain of the trigeminus, irrespective of the branch involved in the pain. 4. The corneal reflex is usually preserved. 5. The approach is bloodless after the dura has been exposed. 6. The operation is much easier and quicker to perform. The operation is, in effect, essentially that of a cordotomy, in that only pain fibers are sacrificed and all forms of sensation are retained. Observations described by Dandy deny the hypothesis that the peripheral branches of the trigeminus are accurately represented by subdivisions of the sensory root. Some postoperative sensory observations suggest that there are separate nerve fibers for various types of sensation. Pain fibers appear to travel separately and to be located exclusively in the posterior part of the sensory root (in cross section). Even when the sensory root is totally divided, varying degrees of sensation are retained in the face. At times, this sensation approaches the normal. This is due to the fact that accessory sensory branches usually accompany the motor root and later join the sensory root. When the accessory branches are absent, anesthesia of the face is complete. The accessory branches of the sensory root apparently never contain pain fibers, nor are pain fibers brought to them by anastomoses with the fibers of the sensory root. The motor root is always preserved because it is at a safe distance from the sensory root. Bilateral tic douloureux can be cured at a single operation by this method because the motor roots are not injured. Occasionally, tumors in the cerebellopontile angle cause tic douloureux. By this approach, they will be disclosed. The chances of a successful removal are enhanced because the tumor is found earlier. When malignant tumors invade the gasserian ganglion, relief cannot be obtained by dividing the sensory root by

the temporal route. The cerebellar route is indispensable in such cases.

Gradenigo's Syndrome: A case presenting this syndrome (suppurative otitis media, severe temporoparietal pain and paresis or paralysis of the abducens nerve) is reported by Garnett Cheney (*J. A. M. A.*, April 6, 1929), in a woman, aged 60, who first came under observation because of severe pain in the left ear and jaw. Three months before, a fiery red lesion on the neck had proceeded by two weeks trouble with some of her teeth which had been crowned by an advertising dentist. Up to the present time, she had continued to have occasional pain in the upper left jaw, but as she was a Christian scientist, she would not permit her husband to call a physician. The left ear was puffy and tender and the teeth were in poor condition, several exuding pus from the sockets. The breath was foul and the tongue coated thickly. The tonsils and throat did not show any evidence of infection. The skin over the lower left side of the face and neck was red and rough. The cervical glands were not enlarged. When the left ear drum was opened, a small amount of thin pus was discharged and considerable relief was felt by the patient. Erysipelas developed the next day, affecting the left side of the face, ear and tongue. It gradually subsided, but during this period, the patient complained of severe pain in the left ear and temple, although the ear was draining well. For four weeks, a dull throbbing pain was present, with attacks of terrific shooting knifelike pains, relieved only by drugs. On the thirty-sixth day of her illness, the patient developed a weakness of the external rectus muscle of the left eye, which persisted for two weeks. Extraction of several abscessed teeth failed to relieve the neuralgia, which persisted for another month, the attacks growing gradually less severe and less frequent. The total course of the illness was three months, although codein had to be resorted to occasionally even after that time. After six months, the patient reported herself free from pain.

Children's Teeth: There are few occasions in dental practice when the practitioner is called upon to exercise judgment, skill and patience to such a high degree as when a child-patient is presented to him. He has to consider not only the condition of the child's mouth at the moment, but even more he has to visualize what will happen to it in the future, according to whether he follows, or abstains from, one or other of several possible lines of treatment. To a much greater extent than in the treatment of the teeth of adults, that of children's teeth makes demands on the dentist for the conscientious exercise of his faculties of observation, diagnosis, prognosis and finally treatment. In no other department of his work can the dentist in a few minutes do so much to make or mar his patient's future.—*Dent. Surgeon*, March 30, 1929.

Anesthesia for Dental Work: Dental cases, besides extractions, include impacted molars and bone cysts. Of the 242 cases in the period considered, 187 were done with gas-oxygen. When ether is chosen it is given intrapharyngeally with a catheter through one nostril, but for gas-oxygen the nasal inhaler is used with good results. Skillful packing of the tongue on the part of the dentist is a decided aid. It not only prevents blood and particles of fillings and teeth from reaching the pharynx, but avoids constant escape and waste of the nitrous oxid and oxygen. For fractures of the jaw it is also desirable to use gas-oxygen given as in dental cases. A choice of gas-oxygen is made so that the patient will be awake at once and have no nausea or almost none. Vomiting with mandible and maxilla wire together can cause aspiration with immediate death from asphyxia or later from pneumonia. We have often given gas-oxygen for jaw fractures with a nasal inhaler, but rather prefer the intrapharyngeal method, as there is less difficulty with mechanical obstruction to respiration.—*Cur. Res. Anes. Anal.*, March-April, 1929.

FOREIGN LITERATURE

Pathologic Histology of the Teeth (Joseph Münch, Fortschr. d. Zahnheilk, No. 4, 1929, p. 258): Abnormal structure of the hard substances, such as hypoplasia, is due to disturbances of calcification, most commonly brought about by rickets. Nutritional animal experiments with diet poor in vitamin D have produced similar varied disturbances in calcification. Müller concluded that it is not the endocrine glands that are damaged in rickets, but that the condition was due to a "dysionie," which is a disturbance of the chemical metabolism in which there is insufficient phosphorus in relation to calcium, and the reaction of the blood becomes acid. It is quite generally agreed that acids and

micro-organisms are responsible for beginning of caries of the enamel, but the exact part each plays, the manner of progress and the variations in the resistance of different teeth are still uncertain. Wild believes that a diet rich in vitamin A is an important factor in combating caries. It is quite certain that an intact outside does not play so important a part in the predisposition to caries as many recent authors of late assume. Seldom does one find caries on extensively abraded occlusal surfaces. The conception that the development of the transparent zone preceding caries of the dentin is a defensive process is being discredited by many. It is presumed to be brought about through disturbances of metabolism induced by caries which brings about a deposition of calcium otherwise held in solution. The development of denticles may often be the cause of neuralgic pains. It is believed that these may produce pain when their development brings about pressure on the nerves of the pulp. Resorption of roots of permanent teeth is very frequently brought about through orthodontic regulation. The more rigid and the more powerful the appliances, the more frequently is resorption produced. Korkhaus believes that this resorption is not due to regulation at an early age but rather to rapidity of movement. Both Ketcham and Korkhaus were against the free use of bodily movement of teeth. The use of a local anesthetic for tooth preparation is harmful. Vacuole degeneration of the odontoblastic layer, hyperemia of the pulp and fibrous degeneration are brought about through its use. Rebel prefers arsenic to procain for pulp extirpation, because arsenic acts as a stimulant to the apical tissue, in consequence of which more regeneration of the hard and soft tissues may be expected. Feldman, on the contrary, on the basis of animal experimentation, cannot corroborate Rebel's findings. He very much questions them and concludes that both methods are inadequate but that the use of procain is the least harmful.

EMIL MUELLER, Chicago.

Internal Secretions (Peter von Kranz, Fortschr. d. Zahnheilk., No. 4, 1929, p. 282): The literature of the past year pertaining to internal secretions is quite extensive. This covers largely experimental laboratory findings that are of great significance to the progress of medicine in general. Comparatively little has been published covering the special field of dentistry. Much of the literature deals with the iodin content of the thyroid under normal and pathologic conditions. The bulk of the literature deals with metabolism as affected by the endocrine glands; vitamins and rickets are discussed in this connection without any reference to the teeth. The pancreas, diabetes and insulin are favorite topics in the discussions of which the words "gingiva" or "tooth" appear not even once. The biologic significance of the ions is gaining ground in spite of the fact that cause and effect is little understood as yet. That limited portion of the literature which deals with dental subjects pertains to pyorrhea, irregular teeth, spotted teeth and tooth retention, as influenced by the internal secretions. Upon the basis of his experiments, Utimuras ascribes internal secretory functions to the parotid and submaxillary glands. All of the dental references are given under literature.

EMIL MUELLER, Chicago.

Pathologic Histology (Herbert von Siegmund, Fortschr. d. Zahnheilk., No. 4, 1929, p. 243.): The adamantinomas are tumors that supposedly develop from the enamel organs of teeth. Of twelve cases encountered in practice four were found in the upper and eight in the lower jaw. While the structure of these is quite well recognized, such questions as to how cysts develop in conjunction with them, or how enamel becomes deposited in them, or, further still, how they develop from the mouth epithelium and the epithelium of the tooth germ are still undetermined. The histologic structure is characteristic and is shown in original photomicrographs. The epithelial cords resemble very closely the epithelium of the normal enamel organ. However, they undergo various types of cystic degeneration; the cells of the epithelium also undergo degeneration, becoming enlarged and dissolved. A complicated picture is offered by the so-called solid adamantinomas. These are more dense and frequently contain areas of calcification. The author prefers to classify the adamantinomas like structures containing enamel or odontomas. The adamantinomas may originate from normal enamel organs, from supernumerary enamel organs, from epithelial rests or from the epithelium of the mouth. These adamantinomas are often confused with tumor-like structures that develop in the craniopharyngeal canal leading to the pituitary body.

EMIL MUELLER, Chicago.

MISCELLANY

THE CLEVELAND CLINIC DISASTER

Through the kindness of John F. Stephan, of Cleveland, we are in receipt of information concerning the Cleveland Clinic disaster in addition to that which appeared in the public press.

The gas was so deadly that although not nearly all the films in the clinic were burned, gas enough to kill 4,000,000 people was generated.

One of the most significant things connected with our profession is the fact that in almost every dental office there are numerous old roentgen-ray films stored away, and while in the nature of things the circumstances which surround roentgenograms in private offices are different from those in a large clinic, the fact remains that great care should be taken in handling such material and a recognition made of the fact that they are dangerous.

When the disaster occurred, there were three men in the dental department, W. J. Peart, C. C. Gilkison, and R. L. Ricksecker. While these men were gassed, the present prospect is that they will all be saved. Dr. Peart is called a hero by the press on account of his presence of mind in helping to rescue a large number, after which he was taken to the hospital badly gassed himself. Dr. Gilkison also on being revived went to work at once to relieve those less fortunate, though he was later sent to the hospital in a very critical condition. It is pleasant to note that the members of our profession acquitted themselves so admirably.

The details of this disaster are unspeakable, and our profession should learn a lesson of caution in the handling of films.

BOOK REVIEW

The Business Side of Dentistry. By Edwin N. Kent, D.M.D., lecturer on conduct of practice, Harvard University Dental School, Boston. Illustrated. 180 pages; price, $5.00. Published by The C. V. Mosby Company, St. Louis.

The great interest that is now being taken in the subject of dental economics will doubtless lead many in the profession to wish to examine this book.

To quote the author, "The present work is the outcome of an insistent demand for an ethical but straight-to-the-point discussion of the problems to be solved in the attainment and maintenance of a profitable dental practice."

In pursuance of this idea, Dr. Kent discusses many topics pertaining to the subject, such as "Dentistry as a Vocation," "Dentistry as a Business," and "The Psychology of Professional Success."

At the present time, there is a widespread interest in dental equipment, and the author under this head remarks: "Dental office equipment, considered as a factor of influence as a business asset, should be viewed from two aspects: first, there is the practical importance of efficient productive machinery, second, the psychological importance of favorable setting."

We are sure that it is the aim of Dr. Kent to recommend the highest ethical aspect of a professional practice, but he makes one statement which might easily be construed otherwise. He says: "A dentist is a business man whether he likes it or not. Being a business men he is subject to the laws, rules, and customs of business life. He may follow a code of professional ethics that will fill a volume and, breaking the standard rules of commerce and finance, he is unethical still."

Probably every reader will know what Dr. Kent means, but if this statement should be interpreted that the ethics of professional life stopped at the point where good business methods were practiced, and there was no ethical obligation beyond this, the result would be unfortunate. We believe that it is the author's theory that there should be no discrepancy between a perfectly ethical attitude and a sane business attitude, and with this we agree; but any tendency to magnify business at the expense of ethics in a professional pursuit we feel very sure would be demoralizing. There is no place in a professional life where balance is more necessary than here, and we should be very careful not

to minimize the importance of ethics in order to magnify that of business.

NEWS

Officers of American Association of Dental Schools: At the annual meeting of the American Association of Dental Schools in Chicago, the following were elected officers for the ensuing year: president, Wallace Seccombe, Toronto, Canada; president elect, Frank T. Breene, Iowa City, Iowa; vice-president, Frederic R. Henshaw, Indianapolis, Ind.; secretary-treasurer, DeLos L. Hill, Atlanta, Ga.

Dr. Bear Dean of Virginia College: Harry Bear, Richmond, Va., vice-president of the American Dental Association, has accepted the deanship of the school of dentistry of the Medical College of Virginia, Richmond, which he will assume July 1. Dr. Bear is at present professor of exodontia and the principles of practice of that institution. He will succeed R. D. Thornton, who has resigned to return to private practice in Toronto, Canada.

Dr. Howe Honored: Percy R. Howe, of Boston, President of the American Dental Association, was the guest of honor at a dinner given by the Iowa State Dental Society at Cedar Rapids, May 8. Roscoe H. Volland, Past President of the Association, was the speaker of the occasion.

Deaths

Cotton, John Ashley, Joplin, Mo.; Western Dental College, 1901; died March 17; aged 52.

Eiser, L. Earl, Chicago, Ill.; University of Illinois, College of Dentistry, 1905; died recently.

English, James Douglass, Worthington, Ind.; died recently; aged 71.

Ford, Herbert A., Los Angeles, Calif.; University of Southern California, College of Dentistry, 1918; died February 8, of heart failure. Dr. Ford served as first lieutenant in the Dental Corps during the World War, enlisting the latter part of 1918, and was a member of the Reserve Corps.

Hines, Harry Christopher, Chicago, Ill.; University of Illinois, College of Dentistry, 1922; died March 18; aged 30.

ANNOUNCEMENTS

CALENDAR OF MEETINGS*

NATIONAL SOCIETIES

American Dental Association, Washington, D. C., Oct. 7-11, 1929.

American Dental Assistants Association, Washington, D. C., Oct. 7-10, 1929.

American Full Denture Society, Washington, D. C., Oct. 1, 1929.

American Society of Orthodontists, Estes Park, Colo., July 15-19, 1929.

Association of American Women Dentists, Washington, D. C., Oct. 7, 1929.

STATE SOCIETIES

District of Columbia, at George Washington University, Washington, D. C., second and fourth Tuesdays in each month from October to June, inclusive.

June (1929)

Colorado, at Colorado Springs (17-19).
Georgia, at Atlanta (12-14).
Maine, at South Poland (20-22).
Michigan, at Detroit (18-20).
Nevada, at Reno, (1).
North Carolina, at Wrightsville Beach (10-12).
Wyoming, at Cheyenne (28-29).

July (1929)

California, at San Francisco (8-12).
Oregon, at San Francisco (8-12).
South Dakota, at Rapid City (1-2).
Utah, at San Francisco (8-12).
Washington, at San Francisco (8-12).
Wisconsin, at Milwaukee (9-11).

American Dental Society of Europe, London, England, July 29, 1929.

Dental Congress of Latin America, Rio de Janeiro, Brazil, July 14-21, 1929.

First District Dental Society of New York, Dec. 10-14, 1929.

*Announcements must be received by the fifth day of the month in order to be published in the forthcoming issue of THE JOURNAL.

International Dental Federation, Utrecht, Holland, Aug. 5-11, 1929.

Pacific Coast Dental Conference, San Francisco, July 8-12, 1929.

MEETINGS OF STATE BOARDS OF DENTAL EXAMINERS

Arizona, at Flagstaff, beginning July 29. Eugene McGuire, Secretary, 420 Security Bldg., Phoenix.

Arkansas, at Little Rock, June 24. Floyd P. Travis, Secretary-Treasurer, Osceola.

Connecticut, at Hartford, June 27-29. Arthur B. Holmes, Recorder, 80 Central Ave., Waterbury.

Florida, at Jacksonville, June 6-8. R. P. Taylor, Secretary-Treasurer, 414 St. James Bldg., Jacksonville.

Indiana, at Indianapolis, June 17. J. M. Hale, Secretary-Treasurer, Mt. Vernon.

Delaware, at Wilmington, June 19-20. W. S. P. Combs, Secretary, Middletown.

District of Columbia, at Washington, week beginning June 17. C. Williard Camalier, Secretary-Treasurer, 406 Medical Science Bldg., Washington.

Kentucky, at Louisville, June 7-11. Robert L. Sprau, Secretary, 970 Baxter Ave., Louisville.

Louisiana, June 20-22. S. F. Gautier, Secretary, 520 Godchaux Bldg., New Orleans.

Maine, at Augusta, June 24-26. G. W. MacKay, Secretary, Millinocket.

Massachusetts, at Boston, June 11-15. W. Henry Grant, Secretary, Room 146, State House, Boston.

Michigan, at Ann Arbor, June 10-15. J. W. Lyons, Secretary, 617 Dwight Bldg., Jackson.

Minnesota, at Minneapolis, June 18. F. E. Cobb, Secretary, 601 Donaldson Bldg., Minneapolis.

Mississippi, at Jackson, June 18. T. Ford Leggett, Secretary-Treasurer, Laurel.

Missouri, at St. Louis, June 5-8. George E. Haigh, Secretary, Central Trust Building, Jefferson City.

Montana, at Helena, July 8-12. T P. Regan, Secretary, Helena.

New Jersey, at Trenton, June 24-29. John C. Forsyth, Secretary, 148 West State St., Trenton.

New Mexico, at Las Cruces, beginning June 13. J. J. Clarke, Secretary, Artesia.

North Carolina, at Raleigh, June 17-20. H. L. Keith, Secretary-Treasurer, 409 Murchison Bldg., Wilmington.

North Dakota, at Fargo, July 9-12. Gilbert Moskau, Secretary-Treasurer, Grand Forks.

Oklahoma, at Oklahoma City, June 24. Charles A. Hess, Secretary-Treasurer, Idabel.

Pennsylvania, at Philadelphia and Pittsburgh, June 27-29. Alexander H. Reynolds, Secretary, 4630 Chester Ave., Philadelphia.

Rhode Island, at Providence, June 25-27. Albert L. Midgley, Secretary, 1108 Union Trust Bldg., Providence.

South Dakota, at Sioux Falls, June 24. G. G. Kimball, Secretary, Mitchell.

Tennessee, at Memphis, June 17. F. W. Meacham, Secretary-Treasurer, 911 Hamilton National Bank Bldg., Chattanooga.

Texas, at Dallas, June 18-21. Maxwell C. Murphey, Secretary, King's Daughters Hospital, Temple.

Utah, June 24-29. S. S. Burnham, Secretary, Thompson Bldg., Salt Lake City.

Vermont, at Montpelier, July 1-3. David Manson, Secretary, Burlington.

Virginia, at Richmond, beginning June 11. John M. Hughes, Secretary, 715 Medical Arts Bldg., Richmond.

West Virginia, at Charleston, June 25-27. C. M. Jividen, Secretary, Charleston.

Wyoming, at Cheyenne, June 25-27. I. J. Hayes, Secretary-Treasurer, Sheridan.

National Association of Dental Examiners, Washington, D. C., October 5. George L. Powers, Secretary, Memphis, Tenn.

MICHIGAN'S SEVENTY-THIRD ANNUAL MEETING

The annual meeting of the Michigan State Dental Society will be held on board the S.S. *Noronic,* leaving Detroit June 18. A three days' cruise through the chain of Great Lakes offers an unusual and novel convention. Visits to the "Soo," Mackinac Island and Lake Superior are included in the itinerary.

Justin D. Towner, Memphis, Tenn.; William J. Gies, New York City; DeForest Davis, Cleveland, Ohio; E. L. Ball, Cincinnati, Ohio, and Howard H. Jackson, Detroit, will give papers. James V. Gentilly and James A. Loughry, Cleveland, Ohio; Carlos H. Schott of Cincinnati, Ohio, and William A. Giffin, Detroit, will give lecture clinics. Several group clinics made up of study clubs from Detroit and other sections of Michigan will add their quota to the program.

Provision has been made to accommodate members of the profession from other states, and their wives.

The *Noronic* leaves Detroit at 9:30 a. m., Tuesday, June 18, and returns to Detroit, Friday morning, June 21. We extend a cordial invitation to all members of the A. D. A. and the Canadian Dental Association and their wives to join us. Reservations may be made by addressing William A. Northrup, 1102 Stroh Bldg., Detroit.

P. C. LOWERY, *President.*

THE MORRIS L. CHAIM AND THE BENJAMIN LORD PRIZE FOR 1929

The First District Dental Society of the State of New York announces a prize of $250 offered by the Morris L. Chaim Fund and a prize of $150 offered by the Benjamin J. Lord Fund, to be known as the Morris L. Chaim Prize and the Benjamin Lord Prize, respectively. These prizes are to be awarded annually at the discretion of the board of directors.

Conditions:

1. Eligibility. Membership in good standing of any bona fide dental, medical or scientific society, or a duly registered student of a recognized educational institution, prior to submission of the manuscript.

2. Date. Papers are to be submitted on or prior to Nov. 1, 1929 to the Secretary of the First District Dental Society, 2 East 103rd Street, New York City.

3. Papers. A. The Morris L. Chaim Prize is offered for the most acceptable paper in the field of science and art as related to dentistry, this paper to embody the results of original research not previously published.

B. The Benjamin Lord Prize is offered for the most acceptable paper in the field of clinical dentistry having an immediate and direct value in its application to practical needs, this paper to embody the results of original research not previously published.

The manuscript shall be typewritten, and accompanied by all necessary photographs, drawings, diagrams and tables, and shall be ready for publication.

The manuscript and all drawings, diagrams, photographs, tables, data, etc., shall be sealed in a plain wrapper or envelop which shall bear on the outside some symbol, group of letters, figures or other identification mark, and accompanying each such sealed packet or envelop, another sealed envelop having on the outside a duplicate of such symbol, group of letters, figures or other mark, and within this sealed envelop shall be placed the name and address of the person submitting the manuscript, etc.

4. Award. The award shall be made by the Board of Directors of the First District Dental Society of the State of New York. At the discretion of the board, the prizes may be divided between the papers adjudged to be of equal merit, or both prizes awarded for one paper, if in the opinion of the judges it merits them.

5. Publication. The First District Dental Society of the State of New York will consider the publication of the successful papers, but publication by the First District Dental Society of the State of New York shall not be binding on either party.

6. Wherever and whenever published, the papers awarded the prizes shall be accompanied by the statement: "Awarded the Morris L. Chaim or the Benjamin Lord Prize, or both, in 1929, by the First District Dental Society of the State of New York."

Further information may be had by addressing: E. M. Davies, General Secretary, First District Dental Society, New York Academy of Medicine, 2 East 103rd Street, New York City.

DECEMBER MEETING FOR BETTER DENTISTRY, FIRST DISTRICT DENTAL SOCIETY, NEW YORK

The First District Dental Society of New York will hold their Fifth Annual December Meeting for Better Dentistry at the Hotel Pennsylvania, December 10-14.

The officers of the society have made arrangements to continue their meetings on the same plan as heretofore and expect that the profession will again support them in making this meeting a success.

JOHN T. HANKS, *Secretary,*
Fifth Ave. and 103rd St.,
New York City

MISSOURI DENTAL BOARD

The Missouri Dental Board will hold an examination in St. Louis at Washington University Dental College, June 5-8. All applications must be filed at the office of the sec-

retary at least ten days before the date of examination. For full information, application blanks, etc., address

GEORGE E. HAIGH, *Secretary,*
Central Trust Bldg.,
Jefferson City

FLORIDA STATE BOARD OF DENTAL EXAMINERS

The Florida State Board of Dental Examiners will hold its annual meeting June 6-8 at Jacksonville, for the purpose of examining all qualified applicants for license to practice dentistry and dental hygiene in the State of Florida. For information and application blanks, write to

R. P. TAYLOR, *Secretary,*
414 St. James Bldg.,
Jacksonville

ASSOCIATION OF AMERICAN WOMEN DENTISTS

The Association of American Women Dentists, organized in 1922 among the women members of the American Dental Association, will hold its annual meeting, October 7, Washington, D. C. Haidee Weeks, Maison Blanche, New Orleans, La., is president, and Wilhelmina Yeretsky, 26 Sheldon Ave., S. E., Grand Rapids, Mich., is secretary.

CELIA RICH, *Chairman,*
Publicity Committee,
Lamburth Bldg.,
Nashville, Tenn.

LOUISIANA STATE BOARD OF DENTISTRY

The Louisiana State Board of Dental Examiners, will hold examinations, June 20-22. For all information, address S. F. Gautier, Secretary, 520 Godchaux Bldg., New Orleans, or Philip M. Gayle, President, Monroe.

SIDNEY F. GAUTIER

NORTHERN ILLINOIS DENTAL SOCIETY

The next meeting of the Northern Illinois Dental Society will be held in Rockford at the new Faust Hotel, October 23-24.

ORVAL A. CROSSAN,
1208 Talcott Bldg.,
Rockford

WYOMING STATE BOARD OF DENTAL EXAMINERS

The Wyoming State Board of Dental Examiners will meet in Cheyenne, June 25-27.

I. P. HAYES, *Sec'y-Treas.,*
Sheridan

NATIONAL ASSOCIATION OF DENTAL EXAMINERS

The next meeting of the National Association of Dental Examiners will be held in Washington, D. C., October 5.

GEORGE L. POWERS, *Secretary,*
Memphis, Tenn.

VERMONT BOARD OF DENTAL EXAMINERS

The Vermont State Board of Dental Examiners will meet July 1-3 at the State House, Montpelier, to examine applicants for a license to practice dentistry and dental hygiene. All applications, accompanied by examination fee, must be in the hands of the secretary at least ten days before the date of the meeting. For applications and other information, address

DAVID MANSON, *Secretary,*
Burlington

UTAH STATE BOARD OF DENTAL EXAMINERS

The Utah State Board of Dental Examiners will hold its next examination June 24-29 inclusive.

S. S. BURNHAM, *Secretary,*
Thompson Bldg.,
Salt Lake City

CORRECTION

In the article by Dr. Ottofy in the May issue, the degree F.A.C.D. was given the author through an error in the editorial office.

PACKAGE LIBRARIES*

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58 E. Washington St., Chicago, Ill.

Alveolectomy.
Amalgam fillings and physical properties of amalgam.
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Best time for orthodontic treatment.
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Cancer of mouth.
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Cavity preparation.
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Children's dentistry.
Cleft lip and palate.
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†Curetting.
Cysts.
†Deciduous teeth and their relation to permanent dentition.
Dental assistants.
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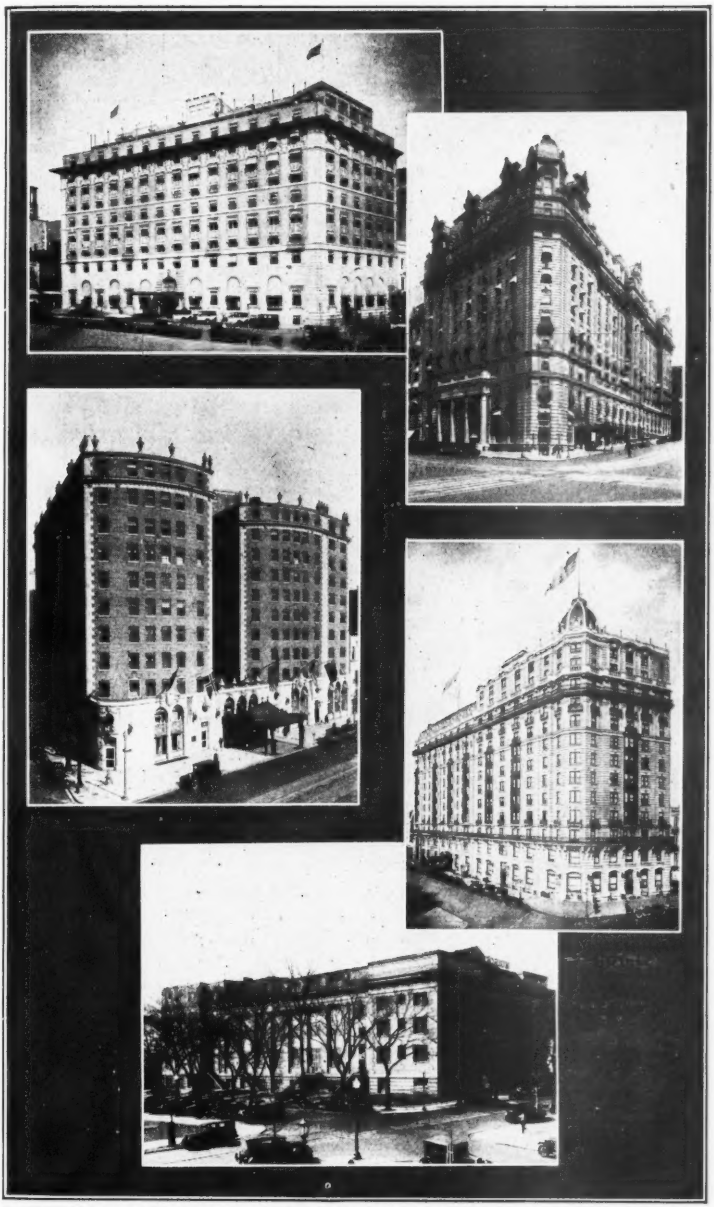
†Dental disease and practice in the tropics.
Dental economics.
†Dental erosion.
Dental ethics.

*Each package library contains information on one of the subjects listed and is composed of reprints, magazine clippings, etc., on that subject only. The packages are loaned to members of the Association for a period of one week after date of receipt. The charge for borrowing a package library is 50 cents. No packages are sold. In response to requests, packages will be compiled on other subjects pertaining to dentistry.

†These packages contain less than the usual number of articles.

Dental hygienists.
†Dental nomenclature.
Dental health education.
Dentifrices.
Dentistry as a career and the status of the dentist.
Denture casting.
Diet and nutrition in relation to teeth.
Diet in pregnancy.
†Dry socket.
†Education of denture patients.
Endocrine glands in relation to teeth.
Esthetics in denture construction.
Ethylene anesthesia.
Etiology of caries.
Etiology of malocclusion and preventive orthodontia.
†Etiology of pyorrhea and its relation to systemic conditions.
Evolution of human dentition.
Examination of the mouth and oral diagnosis.
Extraction of teeth.
First permanent molars.
Fixed and removable bridgework.
Focal infection.
Focal infection in relation to diseases of the eye.
Foreign bodies (of dental origin) in the lungs and esophagus.
Fractures of the jaws.
Full denture construction.
Habits as factors in malocclusion.
†Hemophilia or excessive hemorrhage following extraction.
†High frequency in dentistry.
†Histologic examinations of fractured roots.
†Histologic study of teeth and tissues.
History and progress of dentistry.
History of dentistry.
Hospital dental service.
Impacted teeth and their removal.
Impacted third molars.
Impression technic.
Impressions for inlays.
Indirect inlay technic.
Industrial dental service.
†Infection following extraction of teeth.
†Influence of diet on saliva.
Interpretation of roentgenograms.
Ionic medication.
Lectures to nurses.
Local anesthesia.
†Ludwig's angina.
Mandibular movements.
Materia medica and pharmacology.
Maxillary sinus and antrum—diseases and treatment.
Mouth hygiene.
Muscle exercises and training in orthodontia.
Nitrous oxid-oxygen anesthesia.
Oral diagnosis.
Oral diagnosis with reference to roentgen ray.
Oral manifestations of systemic conditions.

Organized dentistry.
Orthodontic appliances.
Orthodontic education.
Osteomyelitis of the jaws.
Partial dentures.
†Permeability of tooth structures.
Physical therapy in dentistry.
†Pinledge and pinlay attachments.
Porcelain.
Porcelain inlays.
Porcelain jacket crowns.
Postoperative care of dental patients.
Postoperative complications and care.
†Postoperative psychosis.
†Pregnancy and dental disease.
Preoperative examination of patients.
†Preparation of cavities with hypersensitive dentin.
Preventive orthodontia.
Procain (novocain) dermatitis.
†Program building for state and district societies.
Prophylactic odontotomy.
Pulp conservation.
†Pulp nodules.
Pyorrhea.
Relation between dental conditions and mental health of insane.
†Retrusion of the mandible causing impaired hearing.
Root canal treatment.
School dental service.
Sterilization of dental instruments.
Stomatology.
Studies of saliva.
Suggestions for a talk to mothers.
Surgical treatment of pyorrhea.
Tooth arrangement for full dentures.
Toothbrushes and toothbrush containers.
Toothbrushes, tooth pastes and mouth washes.
Traumatic occlusion.
Trigeminal neuralgia.
†Tuberculosis and dentistry.
Ultraviolet ray in the treatment of pyorrhea.
Ultraviolet ray therapy in dentistry.
Use of elevators in extraction.
Value of dental service in schools.
Vincent's infection.



WASHINGTON HOTELS AND (BELOW) THE WASHINGTON AUDITORIUM

HOTELS	Rooms	Single with Bath	Double with Bath	Single without Bath	Double without Bath
BLACKSTONE 17th St., Btw. K. & L.	100	$3.00-5.00	$5.00-6.00		
BURLINGTON Vermont Ave. Thomas Circle	200	3.00-4.00	5.00-7.00	$2.50	$4.00
CAPITOL PARK N. Capitol & E.	150	3.00-4.00	4.00-6.00	2.00 up	3.00 up
CARLTON 16th & K. St.	260	5.00-7.00	8.00-12.00		
COMMODORE N. Capitol & F.	154	2.50-3.50	4.50-6.00		
CONTINENTAL N. Capitol & E.	250	3.00-4.00	5.00-7.00	2.00-2.50	3.00-4.00
DRISCOLL 1st & B. Sts.	200	3.50	6.00	2.00 up	3.00 up
EBBITT 1000 H. St.	150	2.50-3.00	4.00-5.00		
GRACE DODGE N. Capitol & E.	300	3.00-4.00	5.00-8.00	2.50-3.00	4.00-5.00
GRAFTON Conn. Ave., Btw. L. & N.	125	3.50-6.00	5.00-8.00	2.50-3.50	4.00-5.00
HARRINGTON 11th & E.	300	2.50-5.00	5.00-8.00	2.50 up	3.50-5.00
LAFAYETTE 16th & Eye Sts.	200	4.00-6.00	6.00-8.00		
MAYFLOWER Connecticut Ave.	650	5.00-10.00	7.00-15.00		
METROPOLITAN 6th & Pa. Ave.	175	2.50	4.00 up	1.50 up	3.00 up
POWHATAN Pa. Ave. & 18th	300	3.00-5.00	6.00-9.00		
RALEIGH 12th & Pa. Ave.	460	4.00-6.00	5.00-10.00	3.00-4.00	4.00-6.00
WARDMAN PARK Conn. Ave. & Woodley	400	uniform rate $5.00	uniform rate $8.00		
WASHINGTON Pa. Ave. & 15th	400	5.00-8.00	8.00-12.00		
WILLARD Pa. Ave. & 14th	475	5.00-10.00	7.00-15.00		
WINSTON 1st & Pa. Ave.	100	2.50 up	4.00 up	1.50 up	3.00 up

AMERICAN DENTAL ASSOCIATION

Washington, D. C. October 7-11, 1929.

HOTEL RESERVATIONS

In securing hotel reservations for the 1929 Session, consult the hotel rate-sheet and fill out the blank application below. Mail immediately to the hotel you wish to patronize. The hotel will then advise you of the reservation which they make for you.

In case your first choice cannot be made, indicate second and third choices. If none of your choices are available, the hotel manager will mail your application to the Chairman of the Halls and Hotels Committee and he will place your reservation in as favorable a hotel as possible.

Please remember that a reservation constitutes a contract with the hotel to provide you with the accommodations you desire. If you find it impossible to carry out your part of the contract, namely to occupy the room at time agreed on, please write or wire the hotel releasing it, in order that your room may be available for other members.

MAIL THIS APPLICATION DIRECT TO THE HOTEL

HOTEL RESERVATION

AMERICAN DENTAL ASSOCIATION, WASHINGTON, D. C., OCT. 7-11, 1929

.....Hotel. .....1929.
Washington, D. C.

Please reserve sleeping room accommodations as noted below:

.....room(s) with bath for .....people. Rate desired per day $.....

.....room(s) without bath for.....people. Rate desired per day $.....

Second choice hotel.....

Third choice hotel.....

ROOMS TO BE OCCUPIED BY: ADDRESS IN FULL

Applicant..... .....

....

....

....

Please confirm this reservation to applicant. I further agree to notify the hotel at once in the event I am unable to use this reservation.

Important to Hotel Manager: In the event you cannot accept this reservation, please forward this application at once to Dr. R. L. Morrison, Chairman Halls and Hotels Committee, Suite 706, 1103 Vermont Ave., N. W., Washington, D. C., who will attend to the assignment of this reservation.

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1929

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